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NEUROLOGICAL BULLETIN

Clinical Studies of Nervous and Mental Diseases in the Neurological Department of Columbia University

EDITED BY FREDERICK TILNEY, M.D. Ph.D.

VOLUME I

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NEUROLOGICAL BULLETIN

VOLUME I — JANUARY, 1918 — NUMBER I

RECURRENCE OF AN EXTRAMEDULLARY TUMOR AFTER AN INTERVAL OF EIGHT YEARS

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NEW YORK CITY

The following case is of especial interest, not only on account of the nature of the spinal cord tumor itself, but also because of its recurrence after an interval of eight years.

Name: C. T. No. 5596 P. H. Age: 29 years. Nativity: English. Occupation: architect. S. C.: married.

HISTORY. *Present Trouble, Beginning with Complaint.*—In October, 1908, while playing a game of football, the patient was hurt in the scrimmage, immediately after which he felt an intense pain in the region of the spine between the shoulder blades. The pain was so severe that it was impossible for him to remove his clothes without assistance. This pain continued for a week and disturbed his sleep, but after this time gradually subsided. Ten days after this, the patient entered a broad jump contest but, upon attempting to jump, suddenly lost power in both legs and fell. His legs did not become stiff, but he could not move them as rapidly as before. He noticed a numbness in his toes, apparently without any actual loss of sensation in this region.

Gradually his legs became spastic and the numbness increased until September, 1909, when he was admitted to the Presbyterian Hospital, New York City, 11 months after injury. The following notes have been abstracted from his history record while in the hospital.

Previous History.—The patient was, at the age of 21 years, a student in a Southern college. He was active in athletics, playing football and baseball.

In childhood he had measles and typhoid fever at 11 years of age; pneumonia at 15 years, and when 16 years old he had a sore on his penis which lasted one week, but this was not followed by any secondary symptoms. He denies any other venereal diseases and states that his general health has been excellent.

Family History.—His family history as to nervous or mental disease was negative.

VOLUNTARY MOTOR SYSTEM. *Attitudes.*—The patient could walk with great difficulty. Both legs were spastic and weak. Inspection found no sores or other abnormal marks upon his body.

Gait.—His legs were weak and stiff, so that he walked with difficulty, and moved his legs slowly.

Coördination.—No record.

Skilled Acts.—No record.

Reflexes.—The knee jerks were very active. There were bilateral ankle clonus and bilateral Babinski. The cremasteric and abdominal reflexes were difficult to elicit.

Myosthenic Status.—There was weakness in both legs. He could stand and walk with difficulty. There was increased tone of both legs, but no atrophy. He had good control over both arms and the neck muscles. There was no complete paralysis in any part of the body.

General Sensory. *Touch.*—The patient felt the touch of a finger anywhere on legs, back, and abdomen. In some places on the legs and abdomen below the nipple, it was difficult for him to recognize the prick of a pin other than as a touch. In other places the patient experienced pain when pricked with a pin.

Pain.—There was a numbness of both legs, extending up the trunk to a level an inch above the nipple. There were no shooting pains nor gastric crises. There was pain between the shoulders in the region of the original injury, and where he first felt pain after he was hurt. This pain was elicited by jarring or bending forward and had persisted ever since the injury.

Temperature.—He did not recognize heat or cold (presumably in area below line of sensory limit), sometimes calling the hot cold, and the cold hot.

The patient was observed from time to time. The deep reflexes in legs became more active; the sensory disturbances increased until pain or temperature sensibility was more and more impaired.

Supplementary General Examination.—As to rectal and vesical control, his bowels moved daily without catharsis and he had no trouble with his sphincters. For the past five months he had had trouble with starting micturition. Just above the level of the nipple, a little higher on the left side, there was a well-defined band three to four inches wide extending around the body which was distinctly hyperæsthetic. From this level down the sensation was disturbed as just described. The thoracic and abdominal organs and the cranial nerves were all normal, and the arms showed no sensory disturbances or paralysis. The weakness of the legs increased daily. On September 10 and 13, lumbar punctures were made, but no fluid obtained. On September 23 a third lumbar puncture yielded about 20 c.c. of light colored amber fluid, which came out slowly, drop by drop, under low pressure. This fluid showed a marked positive Noguchi reaction and contained very few cells, mostly lymphocytes. During this time his temperature, pulse, and respiration were normal. Urine was normal; Wassermann reaction of the blood negative. There was no tuberculin reaction. Blood was W. B. C. 8,000 (polynuclears, 63%, lymphocytes, 33%, eosinphiles, 2%, basophiles, none); R. B. C. 5,120,000; hemoglobin, 87%.

With the above symptoms, it was inferred that the patient was injured at the time of the scrimmage in football, and that owing to that injury, there was a subdural hemorrhage in the upper dorsal region of the cord and, perhaps, some laceration of the meninges, possibly a fracture of one of the vertebræ, although a radiograph of the spine showed no abnormality. It seemed likely that at the time of the injury there was not sufficient pressure on the spinal cord to cause any decided cord symptoms but that a lesion gradually developed which in some way compressed the cord and slowly increased the symptoms. During the month of October the weakness increased so that the patient was unable to walk. Operation was advised and the patient was sent to the first surgical division on October 27th, and Dr. Joseph A. Blake performed a laminectomy as follows: An incision from the seventh cervical to the fourth dorsal vertebra was made. Considerable bleeding was met with superficially. No evidence of a fracture or dislocation in the vertebræ could be found. The dura, when exposed, showed a little, old, fibrinous exudate. On being opened, the dura was at once expanded bilaterally by a tumor mass which resembled a large blood clot and which surrounded the cord from the seventh cervical to the third dorsal vertebra about 7.5 cm. and showed the consistency of a granuloma. The cord at this point was very much compressed so that it seemed smaller in diameter than above or below but its continuity still existed. The tumor mass was removed piecemeal by a spoon and forceps, care being taken to preserve the cord. After as much as possible of this material was removed, the dura was sutured over the cord with fine sutures. The operation occupied one hour and five minutes. The patient made a fair recovery from the anæsthetic.

Almost immediately after recovering consciousness the patient could move his legs much better than before operation. The temperature after operation rose on the second day to 102 degrees, but immediately fell to normal after the removal of a large bloody exudate which had collected beneath the skin at the point of operation. Catheterization was necessary on the night of operation, but not afterwards.

Subsequent Condition.—The patient's condition steadily improved. He drew up his legs almost to full flexion, which was quite impossible for him to do before operation. The pain and temperature senses had improved so much that they were nearly normal. The ankle clonus, patellar clonus, and Babinski reaction still continued.

November 28th. Improvement continued. The wound was entirely healed. The ankle clonus and patellar clonus were diminished. The patient had been up and walked about with a chair. The patient was gaining complete control over his legs.

December 22nd. The patient left the hospital, walked very well without assistance or without guiding himself with a chair or cane. Sensory disturbance had disappeared. A slight ankle clonus still existed in the right leg. The Babinski reaction was slightly present on both sides. The patient had experienced no more pain between shoulders. He returned to his home in the South. The report received June, 1910, stated that he was then able to walk several miles daily through the woods and was able to play tennis, in fact, that he felt almost as well as ever. In October, 1910, he was still in excellent condition.

The tumor tissue in the gross appeared like a blood clot. Microscopically it consisted of endothelial cells arranged in layers around the blood vessels and lying in a mesh work of fine fibrils. In some places the cells presented a regular star-like radiation outward from a blood vessel. This picture is very much like that of a neuroepithelioma, in which the glia cells and fibre-striations around the blood vessels form stars. In other places the arrangement of the cells appeared more like that in a hemangioendothelioma of the dura. "Throughout the tumor tissue can be seen here and there fresh hemorrhages, and the remains of old hemorrhages; the tumor is, without a doubt an endothelioma of the perithelial type."

Interval History.—After leaving the hospital the patient returned to his home in the South. He was not confined to bed and got about with a cane. There was steady improvement until he could walk without a cane. After nine months he could walk without limping and was able to play tennis, but running was somewhat difficult, owing to weakness of the right foot. Pain in the lower lumbar region persisted three months after leaving the hospital. After two years he felt completely recovered and entered into all his former activities, including athletics. The only symptoms noticed at that time were tiring of the right leg after considerable exercise and occasional slight pain in the lower lumbar region.

Two years after this he was practically well. In the fifth year after leaving the hospital, *i. e.*, four years ago (1913) the patient began to notice an increase in frequency and severity of the lumbar pain, which came mostly at night. This was relieved by lying on his back with a pillow under his lumbar region. Occasionally this pain would come when the patient dived in the water or jumped on the ground landing hard on his heels, and also on bending over a draughting board. Three years ago the patient's pain increased and he began to notice an increasing weakness of the right leg and numbness, gradually spreading from the foot upward. There was no tingling, no formication, no heat or cold sensations, but a "heavy dead feeling."

About two years ago (1915) the left leg became similarly involved and both legs gradually became worse, accompanied with pain in the back which frequently kept him awake at night. The pain was aching in character, never radiating. There was also occasional pain in the right side of the abdomen. No bands of increased or decreased sensation on the body were noted. For the last six months walking has been difficult due to increasing stiffness of legs and clonus when he puts the ball of the foot to the ground. Bowels were normal. Some difficulty in urination was noticed lately on starting the stream. Sexual functions were normal.

On May 17, 1917, the patient re-entered the Presbyterian Hospital, where the following examination was made:

General Examination. *Inspection.*—Scar from the first to the sixth thoracic spine, inclusive. Right leg appears slightly smaller than left. Slight drop foot on right side. Right leg appears to be shorter than the left.

Mensuration.—Not indicated for the arms.

Right anterior superior spine to internal malleolus	89.5 cm.
Left anterior superior spine to internal malleolus	89.5 cm.
Circumference of leg, 15 cm. below patellar region R	30.5 cm.
Circumference of leg, 15 cm. below patellar region L	32.5 cm.
Circumference of thigh, 25 cm. above patellar region R	48 cm.
Circumference of thigh, 25 cm. above patellar region L	49 cm.

Palpation and massage show nothing abnormal.

Voluntary Motor System. *Attitudes.*—Difficulty in standing.

Gait.—When walking there is a general tremor of the body which seems to be caused by clonus in both ankles.

The patient walks in a slow, shuffling manner. The gait is spastic. Difficulty in standing and walking not increased with eyes closed. Special tests, such as walking on toes, on heels, backwards and sidewise, also the tests of Fournier, are performed imperfectly or with great difficulty, because of the spasticity and weakness in the legs.

Coördination.—Equilibratory and non-equilibratory coördination normal, except in so far as the tests were interrupted by the spasticity and weakness in the legs.

Skilled Acts.—Normal. No dyspraxia. Handwriting unimpaired.

Reflexes.—(1 = slight; 2 = active; 3 = very active; 4 = with clonus.) (*a*) Deep.

	Right Left		*Right Left*
Jaw-jerk	1 = 1	Ulnar	2 > 1
Pectoral	1 = 1	Patellar	3 > 3
Biceps	2 > 1	Suprapatellar	3 > 2
Triceps	2 > 1	Achilles	4 = 4
Wrist	2 > 1	Periosteal	2 = 2
Radial	2 > 1	Crossed Periosteal	1 = 1

Clonus

	Right		*Left*
Ankle	4	=	4
Patellar	0		0
Wrist	0		0
Jaw	0		0

(*b*) Superficial Reflexes.

	Right		*Left*
Supraumbilical	2	>	1
Suprapubic	2	>	1
Upper lateral abdominal	2	>	1
Lower lateral abdominal	2	>	1
Cremasteric	1	=	1

A questionable Babinski was observed on the left side; a questionable Chaddock and Oppenheim on both sides. There was no Gordon nor Schaefer.

Myosthenic Status.—Flexion of thighs is weak on both sides, more pronounced on right side. Extension of legs is weak, more so on right. Flexion (plantar) of right foot weak. Dorsal extension of right foot weak.

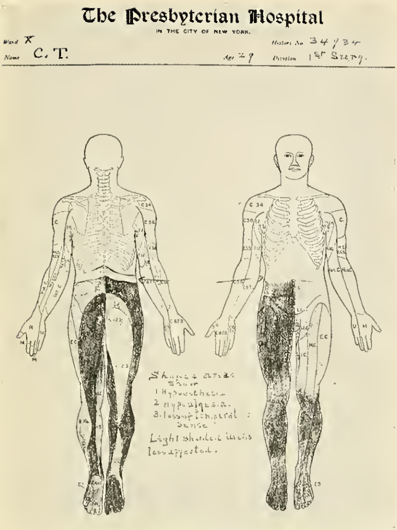
Abnormal Involuntary Movements.—He has no tremors, twitching, spasms, or other involuntary movements.

Muscle Status.—All muscles of the right leg are markedly hypertonic. All muscles of the left leg are slightly so. There is slight contraction in right hamstring muscles and also in right calf muscles.

Abnormal Associated Movements.—Trunk-thigh sign of Babinski is present in both legs. Tibialis sign of Strümpell is present on the right side. No other abnormal associated movements observed.

General Sensory. *Touch.*—Tactile sense is lost in a zone extending down from the level of the fifth lumbar vertebra including the left side of the penis and scrotum. (Fig. 1.) Topognosis over the above area poor; no paræsthesia.

Pain.—Numbness in both legs is evenly distributed; *i. e.*, no patches but a gradual increase from hips downward; more on the right than the left leg. No other form of dysæsthesia except a sensation of disquiet in both legs, so that he must move them constantly, especially the right leg.



The Presbyterian Hospital
IN THE CITY OF NEW YORK.

Ward X
History No. 34734
Name C. T.
Age 29
Division 1st Surg.

Shaded areas show
1 Hypoesthesia
2 hypalgesia.
3. loss of temperal sense
Light shaded areas less affected.

FIG. 1. CHARTS SHOWING AREAS OF SENSORY DISTURBANCE

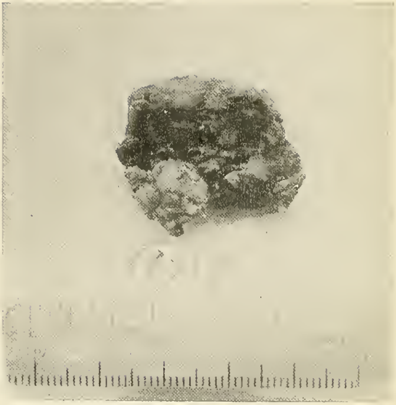


FIG. 2. TUMOR MASS REMOVED
Showing exact size of mass by comparison with ruler below

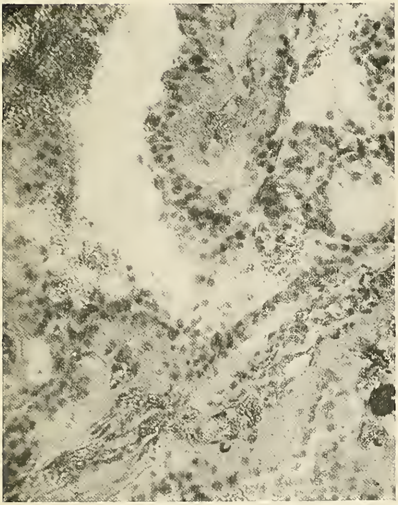


FIG. 3. CROSS-SECTION OF THE TUMOR
Enlargement 150)

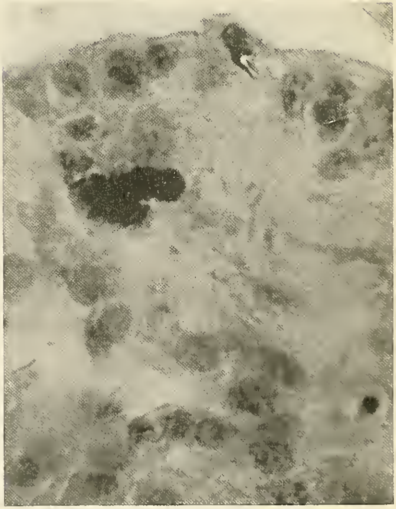


FIG. 4. CROSS-SECTION OF THE TUMOR
(Enlargement 500)

There is pain over the lumbosacral spine over a zone four inches in length, including the area over the fifth lumbar and first sacral, inclusive. This pain, which is constant and dull, is worse at night, is relieved by rest, and increased by exercise.

Pain sense to pin point is greatly diminished or lost in areas shown in figures. The following pain responses were obtained from the galvanic current:

	To elicit pain
4 inches above umbilicus requires	4.5 milliamperes
3 inches above umbilicus requires	3.6 milliamperes
Line of umbilicus requires	5.0 milliamperes
Below umbilicus requires	6.2 milliamperes
Right thigh requires	6.0 milliamperes
Back below T 12 vertebra	5.0 milliamperes
Inner surface right thigh requires	6.0 milliamperes
Back at T 12 vertebra	7.2 milliamperes
Back above T 12 vertebra	4.0 milliamperes

Temperature Sense.—This is greatly diminished over the area noted above and in places is completely lost. Heat sensations are normal over a V-shaped area on back of right thigh and leg and on the right side of scrotum. Temperature sense seems definitely more disturbed than touch or pain.

Vibratory, Pressure, and Muscle-Tendon Sensibility, are all normal. There is no disturbance in posture, weight, or form sensibility.

Myotatic Irritability.—Slight over leg and thigh muscles.

Electrical Reactions.—Faradic nerve excitability: More reaction from same amount of current over anterior crural and anterior tibial nerves of both legs than from stimulation of musculocutaneous nerve of arms. There is more galvanic current needed to produce contraction in leg muscles than in arms. No R. D.

CRANIAL NERVES AND HEAD.—The cranial nerves are all normal, including special sensory, oculomotor, and deglutitional innervations.

MENTAL STATUS.—The mental status is normal.

SYSTEMIC. *Tegumentary.*—The tegumentary status is normal.

Cardio-vascular.—Pulse regular, equal, no increased tension. Blood pressure, S 120, D 62. Heart normal.

Rectal, Vesical, and Sexual Control.—Slight difficulty in beginning urination, otherwise normal; no changes.

LABORATORY TESTS. *Wassermann.*—Negative: spinal fluid, clear, yellow. Globulin: 4+ cells, red cells only; Wassermann on the blood negative. Blood count normal. Urine: glucose present on normal diet. Sugar free on diet without bread or sugar.

SUMMARY.—Spastic diplegia. Anæsthesia of lower extremities and lower part of back and abdomen.

DIAGNOSIS. *Spinal Cord Tumor.*—By means of faradic stimulation, Dr. Tilney finds a level of diminished sensibility to fine touch to a level of the twelfth thoracic spinous process behind and coming around two inches above umbilicus. He puts the level of the mass at the ninth and tenth thoracic segments.

The patient was operated on June 9, 1917, a laminectomy being performed. A median incision was made down to the spines of the vertebræ and the muscles were separated from the spinous processes and laminæ. The laminæ and spinous processes were removed by rongeur forceps. The dura was opened and a diffuse tumor was found extending for the entire distance of the opening and reaching above the opening and below it. The tumor was purple in appearance and irregular in shape, appearing larger in some portions than in others. It was very vascular and completely surrounded the nerve roots. It appeared to be in great measure behind the cord. Several pieces were removed for examination, with considerable hemorrhage. The dura was left open. The muscles were sutured over the tumor and open dural sac. The skin was sutured with silk worm gut and silk.

On microscopic examination the tumor was found to be made of a large number of small blood vessels, the walls of which were thickened by a hyaline degeneration. Surrounding these vessels and forming the interstitial tissue between them were masses of round mononuclear cells with a deep staining nucleus about the size of a large lymphocyte apparently of mesoblastic origin.

The tumor resembled the one formerly removed except for the hyaline degeneration of the vessel wall. It might be considered either as an angiosarcoma or perithelioma, but was given the diagnosis of endothelioma.

Pathological Report.—The specimen of the tumor consisted of a flattened mass of tissue about 23 cm. in diameter which was blood-stained and friable and seemed to be composed of pinkish lymph glandular-like tissue through which were innumerable tiny blood-stained areas.

June 23, 1917.—The patient was transferred to the Medical Division. The wound on the back was healed completely. The patient was able to move his legs much better than before operation, but they were still weak. He complains of tingling in the legs but no persistent clonus as before. Ankle clonus can be brought out, but before operation the simple voluntary act of raising the leg brought on a clonus which has disappeared since operation. The patient is free from pain and quite comfortable. Beginning urination is a little retarded, but no incontinence exists. Defection is normal.

The patient is not aware of anæsthesia but believes his lower extremities are much less hypæsthetic than before.

He is sugar free. Carbohydrates are being steadily increased.

He is receiving general massage which makes him feel much better.

July 24th.—Hypæsthesia still present below the original line but very much less marked than before the operation. Left side is almost clear. Sensation is restored on penis but is absent over both sides of scrotum. There is marked improvement on the legs and feet.

Hypalgesia corresponds to hypæsthesia. Temperature sense is still the most disturbed and diminution is generally present over the whole of the right lower extremity and left leg.

The feet seem to have cleared up more quickly than the legs or thighs.

There is no spasticity, no permanent clonus, no Kernig, no Babinski or modification thereof; the reflexes (deep) are very active.

The patient has steadily gained strength; he can support his weight on his legs and walk with aid of a chair for support. No clonus, no anæsthesia exist, and general condition is good. In August he left the hospital for his home in Tennessee.

From the records of the first operation it is evident that all the growth was not removed at that time, as it was removed piecemeal. The improvement was slow but not unduly so for such a complete degree of pressure. The growth undoubtedly increased slowly up and down the canal but gave rise to no symptoms for four years. It is possible that it did not grow for some time after the operation and then suddenly took on a rapid growth. This does not seem probable, however. We believe that the tardiness in the appearance of the symptoms may be explained by the soft nature of the growth and its vascular character.

At the last operation we found that the level of maximum pressure was not at the upper limit of the growth, in fact, was well down below the level. It was such a soft growth that there were no root pains although the nerves were completely surrounded and embedded in the tumor mass. The tenderness which was present over the spinous process at the site of the greatest pressure is comparable to that which is seen from time to time in cases of spinal cord tumors and is best explained by involvement or pressure on the dorsal branch which supplies dura and periosteum of bone at this level. It did not invade the nerve tissue.

The complete removal of the tumor was quite impossible. Bleeding was most profuse on every attempt, and we contented ourselves with the removal of a portion for histological examination. (Figs. 2, 3 and 4.) The dura was left open to afford room for growth without exerting pressure on the cord. It was thought that a real improvement, which would exist over a long period, might be obtained in this way as the tumor was slow-growing, having slight powers for compression and no tendency to invade the nervous tissue.

The course of the patient's case subsequent to leaving the hospital has amply justified such a procedure, as his improvement has been continuous and he now has completely regained the power of his legs. Endotheliomata vary greatly in their malignancy, but this one certainly has shown no tendency to metastasis and has given rise to no cachexia. It is much too soon to give any accurate prognosis but it would seem as though the ultimate outlook were bad.

POLIOMYELITIS WITH PROLONGED SOMNOLENCE

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This case is brought before the conference because it presents an unusual complication of infantile paralysis. The boy at present is four years of age and at this time the complaints, made by the mother, are as follows:

1. Wasting of right leg. 2. Dragging of right foot. 3. Weakness of right leg, particularly when walking or running, and especially, if walking any distance.

PRESENT ILLNESS, BEGINNING WITH COMPLAINT.—The history of his present trouble is as follows: On September 22, 1916, he left New York for Liverpool in good health, remaining so for the entire trip across the ocean; he was not even seasick. Upon arriving in Liverpool on the 29th of September, he said, "I don't feel well; please carry me. Please take me to a doctor." This speech, so unusual for a child of his age, indicates that he must have been ill. The mother carried him all day. He seemed weak, but had no pain and his body did not seem warm. He refused his bottle and at 7 p. m. was still complaining, repeatedly asking to be taken to a doctor. That evening he was seen by a physician who looked at his tongue but made no further examination. On learning he had just arrived from New York, the doctor said: "Probably infantile paralysis."

Another physician was called the next day who found the child sleeping, in which condition he remained, not even arousing to take food. There was apparently no temperature. On the fourth day he was still sleeping. The mother then insisted on a consultation as a result of which, however, no diagnosis was offered but the recommendation was made that the baby be sent to a hospital for further study. This suggestion was followed, but the child still remained in a profound sleep. It was impossible to awaken him and he did not take nourishment. The mother herself took the child to the hospital and laid him in bed upon his right side. Each time she called to see him he still lay on his right side in the same posture, apparently unchanged even when she called six days later to take him away. She was informed that he had taken no food during all this time.

After fourteen days, the boy made the first sound, calling his mother. She then took him to London. He was there seen by a physician who thought the trouble had been a spinal meningitis. The child was unable to hold up his head without assistance for six months. He sat up alone for the first time on the first of February, 1917; two weeks later he was able to stand; and has since been gradually improving, the right leg remaining much thinner and softer than the left.

The neurological examination showed the following conditions:

VOLUNTARY MOTOR SYSTEM. *Attitude.*—No abnormal attitudes were observed.

Gait.—The locomotion of the child was defective. The phase of progression in the left leg was normal, that in the right leg showed an inability to produce the proper amount of normal flexion of the foot as well as insufficient flexion at the knee and hip. The heel–toe time element was distinctly disturbed so that heel and toe of the right foot came in contact with the floor at about the same time, there being no appreciable interval as is the normal condition. The phase of support upon the left leg was normal, that upon the right leg showed a loss of power in the gastrocnemius-soleus group as well as in the hamstrings, so that there was a well marked knee-back. These defects resulted in a slight limp in which the patient favored the right leg.

Coördination.—No ataxia was observed in any of the extremities or in any other part of the body, equilibratory and non-equilibratory coördination being normal.

Skilled Acts.—All skilled acts were normal except for a slight defect in the movements of the right leg.

Reflexes.—The deep reflexes in both upper and lower extremities were about equal on the two sides. There was no appreciable difference between the patellar and Achilles upon the right and the left. The superficial reflexes were normal and no pathological reflexes of any kind were observed.

Abnormal Involuntary Movements.—No such motor disturbances were present.

Muscle Strength.—There was a distinct loss of power in the right leg as compared with the left in the following groups: Quadriceps extensor, gastrocnemius, soleus, and anterior tibial group. Otherwise, the musculature developed normal strength, both to resistance and in the performance of definite acts.

Muscle Status.—There was a noticeable loss of tone in the quadriceps extensor group, also in the gastrocnemius, soleus, and anterior tibial groups of muscles on the right side. A slight atrophy was apparent in the thigh muscles on the right side with some loss of contour, the flattening being confined mostly to the quadriceps extensor region. A similar flattening was observed over the anterior tibial group.

The electrical reactions showed no alteration in the normal contraction formula, cathode being greater than anode with a tendency of the muscles to manifest a hyperexcitability to the galvanic current of all strengths. The muscles of the right as well as of the left leg reacted to both galvanic and faradic stimulation in a normal manner.

Abnormal Associated Movements.—No such movements were observed in any part of the body.

NERVE STATUS.—Nothing abnormal was observed which indicated any pathological condition in the neural status.

GENERAL SENSORY.—Stimuli for *touch*, *pain*, *temperature*, *vibratory*, *pressure*, *muscle-tendon*, *stereognostic*, and *barognostic sensibility*, all gave normal responses throughout the entire body.

CRANIAL NERVES AND THE HEAD.—All of the cranial nerves were normal including the olfactory, the optic, the oculomotor, trochlear, and abducens, the trigeminal and facial, the acoustic in its two divisions, cochlear and vestibular, the glossopharyngeal, pneumogastric, spinal accessory, and hypoglossal nerves.

MENTAL STATUS.—Nothing abnormal was noted in the mental condition of the child.

CRANIAL MORPHOLOGY.—There were no abnormal appearances or measurements with reference to the head.

SYSTEMIC EXAMINATION. — *Tegumentary*, *glandular*, *cardio-vascular*, *respiratory*, *gastro-intestinal*, *genito-urinary*, *and skeletal* systems were normal.

No laboratory tests or *X*-rays were made in the case.

The question is one of diagnosis. The physician who first saw the case and the one who saw it later gave the two possibilities, namely poliomyelitis and spinal meningitis. So far as onset goes, it was of course characteristic of both of them. So far as the history goes, it was atypical for both, though more typical of poliomyelitis. The absence of temperature makes it seem unlikely that we are dealing with a meningitic process. Absence of temperature in poliomyelitis is a common observation, or, if there is any, it is of such short duration as to pass undetected. The distribution of motor disturbance is that of poliomyelitis rather than meningitis. The indications are that we are dealing with flaccid disturbance, for from the first the muscles were relaxed, indeed, we have good evidence in the history and in the present status of flaccid conditions. As for visceral disturbance, he was at the time, as these cases usually are, incontinent.

The prolonged stupor is unusual in poliomyelitis. For fourteen days he remained almost immobile and practically comatose, taking no food during this time. This is a strong argument against poliomyelitis as we ordinarily know it. On the other hand, poliomyelitis which affects more than the cord, involving the brain as well, a polioencephalitis, does show marked cerebral changes. The cases with which I am most familiar, however, have shown a tendency toward excitation or even delirium.

DISCUSSION

DR. STRONG.—The uvula deviates to the left and I thought there was a slight facial paralysis which would indicate brain involvement.

DR. CHARLES A. MCKENDREE.—In his presentation of this case, Dr. Tilney has left little to be said in addition. The history is certainly remarkable. According to the anatomical findings, the case appears to be definitely one of poliomyelitis with possibly some cerebral involvement. But it seems to me most unusual that there should have been such extensive involvement of the cord and brain without irritation phenomena, more active meningeal symptoms and temperature. The paralysis, which was apparent after emergence from the stupor, was extensive and seems to have left behind fairly noticeable changes. In such a case, particularly, it is regrettable that we have no record of the spinal fluid and blood findings which would have been of great value in making the diagnosis at the time of onset. The case seems to be one of atypical polioencephalitis.

DR. TILNEY.—We have in the medical handling of this case quite striking evidence of the effect of the war on medicine in England. England has been stripped of medical men and most of the attention of those remaining at the present time is devoted, not to the civilian population, but to the army. To that we may probably attribute the fact that we have no record of blood culture or spinal fluid findings.

DR. HOYT.—I, too, believe the case to be one of polioencephalitis, but I think for the sake of completeness we should consider the possibility of poisoning, although I do not recall the drug or food protein that would act in that way. There was no temperature, the child had been for longer than the incubation period on a ship where there was no other case of poliomyelitis, and in an environment where there was plenty of chance of poisoning by food. I would like to hear if any one knows of any drug or food poison that would produce this profound and prolonged somnolence.

DR. HALLOCK.—It is doubtful if the report of the case is accurate; I mean in regard to whether there was temperature or not. It is very unlikely, with a history like this, that the child had no temperature. The mother may have been told this to reassure her and she may not have discovered it herself.

DR. GOODHART.—I think the very complete presentation of this case, in conjunction with the observations of the last three speakers, suggests very strongly the fact that this is one of those anomalies seen in our clinical observation of the cases and classes of poliomyelitis. To my mind, assuming that the child had no temperature and the observations at the time were as reported, we are evidently dealing with poliomyelitis, in a sense pure and simple but with manifestations of encephalitis during the toxic involvement of the brain, whereas the active pathological process deals principally with the spinal cord. I don't think you can in any other way explain why there was an overwhelming of consciousness with no evidence of active meningeal involvement or active encephalitis, the process being entirely or almost entirely localized in the cord.

DR. TILNEY.—Considering Dr. Howe's work on the last epidemic, perhaps he can throw some light on the cerebral changes.

DR. H. S. HOWE.—We did not find marked cerebral changes in any of our cases. There was frequently some evidence of meningitis, but this was never of the same degree as that seen over the cord and cerebellum. It was really more of an edema with very little cellular infiltration or fibrin formation. It seems quite probable that an edema of the cerebral pia might cause a prolonged state of coma from which the patient would recover without cortical lesions, as was seen in this case. In the experimental animals there were usually more signs of inflammation in the cerebral meninges, but here the inoculation had been intracerebral.

DR. MICHAEL OSNATO.—Along the lines which Dr. Hoyt suggested, at the same time that the poliomyelitis epidemic occurred last summer, there was an epidemic in Boston of acidosis, thousands of cases occurring in young children, and the symptoms are brought to my mind by this particular case, except possibly for the lower motor neuron findings. Some of these children remained stuporous for a number of weeks. Many died, but those who recovered showed great wasting, not always evenly distributed throughout the body. This case is atypical to the extent in which recovery has taken place, even for poliomyelitis. The electrical reaction was an essentially normal one. The fact that this child had no temperature is a point against poliomyelitis. In a case where the infection was so profound, in the beginning particularly, temperature must have been present if it were poliomyelitis. Draper reports a number of cases in which, in the post-paralytic stage or soon after paralysis, it was found that at the time the child had no temperature, but it was ascertained that a few days before there had been temperature. According to the history, this child had no temperature at any time. I have a suggestion to make in this case. If the mother would consent, it might be a good idea to get some of this child's serum and see what effect it would have on an infected monkey. That would confirm the diagnosis.

Dr. Tilney.—We do not know positively whether there was temperature or not before the appearance of the symptoms. That point was covered by the speaker who said the temperature might have been present but the fact concealed from the mother.

Dr. George A. Blakeslee.—The atypical part of the case is the profound somnolence. Many poliomyelitis cases have that for a few days, but not for two weeks. After such stupor there are usually no manifestations of cranial nerve involvement. There is some involvement here, but in the many cases with these stupors there is no such involvement. Physical examination shows hypotonia and diminished reflexes, showing a lower motor neuron involvement. We have had cases with similar histories, with little fever. From my experience in Bellevue last summer, during the acute stage of the disease, there were many cases which on palpation you would not think had fever, but the rectal temperature showed 100 degrees to 100.5 degrees. We find there is a loss of muscle tone; the only way to determine the exact condition of the muscles, whether there is a diminished excitability or a degeneration, is to carefully examine each individual muscle with faradic and galvanic electricity.

Dr. John H. Nolan.—I do not know of any food or drug poisoning which would cause these symptoms. In regard to the condition being due to an acidosis: This illness is most commonly present between the ages of one to three years and is characterized by vomiting and coma. The mortality is about eighty-five to ninety per cent. No child could survive an acidosis of three weeks' duration. My experience with polioencephalitis has been limited, but I think it is most unusual to see a case without generalized or localized convulsions, also with special senses affected, and the recovery of my cases was never so complete. The mentality, too, is often impaired. I would like to ask the mother: Did the illness begin with convulsions? What was the first thing she noticed? (Just sleeping.) Was his vision affected? (He noticed nothing so she could not say. He did not answer when she tried to arouse him.) I consider the case one of polioencephalitis with toxic meningitis which has cleared up.

A DISCUSSION OF THE SUBJECT OF APHASIA WITH A CLINICAL REPORT OF THREE CASES

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The first case bears the official conference diagnosis of motor aphasia, right hemiplegia (descriptive diagnosis), thrombosis (etiological diagnosis), anatomical diagnosis thrombosis, in a branch of the middle cerebral artery leading to destructive changes in the posterior limb of the internal capsule. In the second case the official diagnosis is paraphasia, right hemiplegia, embolism in the posterior limb of the internal capsule.

CASE I.—Patient: J. M.; male; age 36.

HISTORY. *Present Illness, Beginning with Complaint.*—On the night of December 16, 1916, this patient retired apparently in perfect health and awoke the following morning complaining of weakness in the right arm. There had been, prior to this, no headache, no dizziness and no numbness, and the weakness in the right arm was not accompanied by any of these symptoms. Later in the day the patient became drowsy and finally unconscious. His wife found him on the floor with paralysis of the right half of the body; the patient's eyes were open and he seemed to recognize her, but he could not speak. The wife stated that she had been gone not more than one hour and that when she left her husband was not unconscious. The patient stated, however, that he was unconscious during this time, but we have no positive information upon this point. We are certain, however, that he was not unconscious more than one hour, if at all.

Previous History.—On March 19, 1916, the patient was struck by a street car and suffered severe contusions of the right side of the face and of the region about the right ear. Immediately after this injury he complained of defective hearing in both ears, more severe in the right. Following the injury, there was no hemorrhage from the nose, ears, or into the eyes, and it was said at the hospital when the patient came for observation that there was no fracture of the skull.

The patient has led a very active life. He was at one time in the British army, and has been all over the world, most of the time in tropical climates. He has been a very hard drinker. Venereal infection is denied.

In order to understand the aphasic status in this case, it is necessary to know that the patient is practically illiterate and that he could read and write very little before the injury. He was able to read simple articles in the daily newspaper with difficulty and could write only the simplest of letters. There is a congenital cataract of the right eye. On inspection the patient was seen to be thin and poorly nourished.

Family History.—The family history is not definitely obtainable.

VOLUNTARY MOTOR SYSTEM. *Attitudes.*—No record.

Gait.—The patient walks with a distinctly hemiplegic gait, the right side being affected.

Coördination.—Is not defective.

Skilled Acts.—Speech. The aphasic status was determined by the following series of fifteen questions suggested by Professor Tilney. These questions and respective responses enable us to investigate and to draw conclusions concerning the skilled act of speech. The interrogations are as follows:

Question 1.—Can the patient speak spontaneously in ordinary conversation? (What is your trouble?)

Answer.—The patient cannot answer this question.

Question 2.—Can he enumerate, denominate, and name objects?

(Can you count your fingers aloud?

Can you name the members of your family or the days of the week? Can you name these objects?)

Answer.—These questions are answered well, although there is a slight defect in the naming of the members of his family and the days of the week.

Question 3.—Can he speak from dictation?

Answer.—Very imperfectly.

Question 4.—Can he read aloud?

Answer.—Cannot.

Question 5.—Can he read to himself and understand what he reads?

Answer.—This the patient cannot do at all well. In fact, this answer must be considered an absolute failure.

Question 6.—Can he recite what he reads; can he say his prayers?

Answer.—This reply is also a failure; the patient cannot repeat what he has read, cannot say his prayers aloud and cannot sing a song aloud. He carries a tune perfectly well and gets many phrases in the song, but cannot be said to sing the words coherently and intelligibly.

Question 7.—Can he write spontaneously?

Answer.—In answer to this question, the only thing the patient can do is to write his own name, and very frequently only his last name.

Question 8.—Can he enumerate and denominate in writing; write the numbers from 1 to 10; add a column of figures and name objects shown?

Answer.—The patient can write from 1 to 10, but cannot add, cannot write the days of the week, and cannot write the names of objects shown to him.

Question 9.—Can he write from dictation?

Answer.—The patient cannot write from dictation. In considering these last two tests it must be remembered that this patient is possessed of a very poor quantity and quality of education and some allowance must be made for this.

Question 10.—Can he copy written or printed matter?

Answer.—The patient must be said to fail in doing this test but can copy his own name and that of his wife, but very little else.

Question 11.—Can he hear and understand what he hears?

Answer.—The patient hears and understand, what he hears perfectly. He recognizes the watch when placed close to the left ears the right one being deaf. The patient's eyes were closed in the watch test. He obeys very well all spoken commands, providing they are not too complicated.

Question 12.—Can he see and understand what he sees?

Answer.—This is well done.

Question 13.—Is he aware of errors made in speech or writing?

Answer.—The patient is well aware that he makes mistakes in speaking and writing.

Question 14.—Has he a concept of his speech? (Lichtheim's Test.)

Answer.—The patient's idea about his difficulty is that he understands everything and has the mental picture of what he wants but cannot say it. Lichtheim's test could not be done on this patient because of lack of education. He does not know what syllables are and therefore naturally could not be expected to perform this test properly, although he is able to tell the number of letters in most words, particularly words and names with which he was familiar before the onset of his trouble. In general, we may say that the patient has a correct conception of his speech.

Question 15.—Does he misuse words? Is he ungrammatical out of proportion to his education? Does he perseverate? Does he speak a jargon?

Answer.—The patient occasionally misuses what few words he does utter, but speaks a jargon and is prone to repeat.

In checking off the list of questions, we find that the patient fails in the proper response to the questions asked in numbers 1, 3, 4, 5, 6, 7, 8, 9, 10, and 15. Considering Lichtheim's test as properly done we may conclude that we have a subcortical aphasia, very nearly purely motor in type.

Reflexes. Deep and superficial.—All pupillary reactions are normal. The right triceps, biceps, patellar, and ankle reflexes are 3 plus. The corresponding reflexes of the left side may be expressed as 1 plus. The right abdominal and epigastric reflexes are absent, but the cremasterics are equal and active. There is a Babinski, a Chaddock, and Oppenheim on the right side, and a fairly well marked clonus, which, at times, is easily exhaustible.

Abnormal Involuntary Movements.—No record.

Muscle Strength.—Is 3 minus in the muscles of the right arm, shoulder and leg, and 2 plus in the right lower and middle facial muscles.

Muscle Status.—The atrophy in the right shoulder girdle muscles is very considerable, but the *electrical reactions* do not show the reaction of degeneration.

Abnormal Associated Movements.—Such as ordinarily appear with fairly well marked hemiplegias are present in this case, but there are no athetoid or choreiform movements.

NERVE STATUS.—There is no record.

GENERAL SENSORY.—There are no sensory changes.

CRANIAL NERVES AND HEAD. *Acoustic and Ear.*—Hearing is defective in slight degree to air conduction on the left side, but very markedly involved on the right side.

Bone Conduction.—Bone conduction is normal on both sides.

Hypoglossus and Tongue.—At this time there is only slight deviation of the tongue, although several months ago, there was marked deviation of the protruded tongue, to the right.

MENTAL STATUS.—The patient has become extremely emotional, crying occasionally though usually a prey to immoderate and ill-timed outbursts of laughter or rage. He may be said to be extremely irritable.

PHYSICAL EXAMINATION. Heart and lungs are negative. The pulse is 70, temperature normal. There are no palpable glands and the blood pressure is 160 systolic and 110 diastolic.

LABORATORY TESTS. *Wassermann.*—The blood Wassermann was negative on several examinations.

Urine.—The urine is negative.

Special.—The fundi are normal, but the arteries in the retina are thin and tortuous.

HISTORY AFTER THE ONSET.—Immediately after the onset this patient was practically wordless, the only thing he could say was "Sure!" in an explosive manner. This word he used in answer to everything. Almost exactly one month after the onset he called his wife by name, then his niece, and he has gradually used the names of many of his friends and relatives. From the wife it has since been learned that the patient had pneumonia three years ago and a rather acute and prolonged attack of rheumatism about half a year before that.

CASE II; age 19; single; a marble worker by occupation.

HISTORY. *Present Illness, Beginning with Complaint.*—The patient had bronchopneumonia three years ago. The left iris was operated on for cataract when he was a baby. Aside from these two incidents, the patient had been perfectly well until April 14, 1917, when he was operated on for appendicitis at St. Catherine's Hospital, Brooklyn. Three days later he became paralyzed in the right face and arm and leg and was unable to speak. About two years ago the boy is said to have had his first epileptic seizure. The attack was very severe and was repeated about one year ago. These two epileptiform convulsions were not followed by speech defects or by paralysis, and have not been repeated to date.

Previous History.—It is of interest in considering the aphasic status of this patient to know that he went to school at six, attending the public school until he was sixteen years of age; when he left he was still one year short of graduation. This apparent backwardness was due to irregular attendance and partly because of three operations for cataract on his left eye; this kept him out of school at various times almost continuously for three or four months. Aside from this, however, the boy was a truant and was unmanageable in so far as his attendance to school duties was concerned. He was always bright and keen and picked things up easily, read a great deal of boys' fiction and newspapers, but cared for no serious literature. He wrote a fine hand and was able to compose a fair letter in writing to his friends and relatives. He spoke Italian fairly well, but English was always the language of choice and was spoken and written much better than Italian. In fact, he never could write Italian, and, of course, cannot do so now.

Four years ago he began to work in a shoe factory where he worked for three months, and then went to a marble works for seven months, and back to the shoe factory until recently. In fact, this boy can be said to have had the average education of a boy who leaves school in the grade known as 7-A; the average age for these children is twelve. It will be remembered that he was sixteen when he left.

Not only this.patient, but the patient above reported, have always been right-handed and have come from right-handed families. They both also favor their right feet in performing skilled acts with these extremities. On inspection the patient was found to be a well-nourished individual who presented no abnormalities excepting the right hemiplegia.

Family History.—The patient is the oldest of eight healthy children, and the father and mother are both living and well. The mother had two non-induced miscarriages.

VOLUNTARY MOTOR SYSTEM. *Attitudes.*—No record.

Gait.—Hemiplegic.

Coördination.—No record.

Skilled Acts.—Skilled acts, particularly of speech, are defectively performed, as is shown by the following aphasic status of the patient given in exactly the same way as in Case No. 1.

Question 1.—Can the patient speak spontaneously in ordinary conversation?
(What is your name?)

Answer.—The patient cannot speak spontaneously in ordinary conversation.

Question 2.—Can he enumerate, denominate, and name objects?
(Can you count your fingers aloud? Can you name the members of your family or the days of the week? Can you name these objects?)

Answer.—Can be done with a defect. The defects appear to be mostly in naming objects.

Question 3.—Can he speak from dictation?

Answer.—He cannot speak from dictation.

Question 4.—Can he read aloud?

Answer.—He cannot read aloud.

Question 5.—Can he read to himself and understand what he reads?

Answer.—He can read to himself and seems to understand what he reads.

Question 6.—Can he recite what he reads; can he say his prayers?

Answer.—The patient cannot repeat aloud what he has read, cannot say his prayers aloud, but he can sing 'My Country 'Tis of Thee' all through without a mistake in the words, but when asked to repeat the words of the song, he cannot do it.

Question 7.—Can he write spontaneously?

Answer.—The patient can write spontaneously but uses wrong words. For instance, when asked the question: "Please write down what troubles you have," he puts down the phrase, "I am England." When asked what this means, he replies, "I had appendicitis." When asked to write my name, he wrote, "Dr. Fris."

Question 8.—Can he enumerate and denominate in writing; write numbers from 1 to 10; add a column of figures and name objects shown?

Answer.—The patient can write the numbers from 1 to 10. He can add up simple columns of figures and can write the days of the week and can also write the names of various objects which were shown to him, but will often make mistakes which he very often corrects himself.

Question 9.—Can he write from dictation?

Answer.—The patient can write only simple things from dictation but leaves out letters and quite often words, and in Question 10 he will do the same thing; *i. e.*, he can copy form print but will omit words or letters.

(Question 10.—Can he copy written or printed matter?)

Question 11.—Can he hear and understand what he hears?

Answer.—The patient hears and understands what he hears, but not on all occasions.

Question 12.—Or sees?

Answer.—The patient sees and understands what he sees. The following peculiar reaction should be noted. I asked him to look at my watch and then asked him what time it was. He could not tell me in words nor could he write it, but he drew a diagram of a watch on the blackboard with thirteen numbers on it, there being 2 tens, and then drew the hands of the watch, so that the result of the diagram presented the correct time.

Question 13.—Is he aware of errors made in speech or writing?

Answer.—The patient is aware of errors in both speaking and writing.

Question 14.—Has he a concept of his speech? (Lichtheim's Test.)

Answer.—The patient is able to tell the number of letters in a word, but does not seem able to tell the number of syllables, although he assures me he understands what syllables are. Later after a long explanation he finally demonstrated that Lichtheim's test is positive.

Question 15.—Does he misuse words? Is he ungrammatical out of proportion to his education? Does he perseverate? Does he speak a jargon?

Answer.—The patient is practically wordless. Therefore no ungrammatical uses of words could be made out. Whatever utterances he has, and they are very few indeed, he perseverates.

Summing up this case, therefore, we find a defect in 15, 10, 9, which are only partial defects, a partial defect also in 8, a total defect in 7, a defect in 6, 4, 3, a partial defect in 5, a total defect in 1. In other words, the patient practically has a defect in the performance of the entire list, although he is better in some than he is in others.

Reflexes.—All the pupillary reflexes are normal. The right triceps, biceps, and patellar are all greatly exaggerated and can be indicated as 3 plus, the left as 1 plus. The right abdominal, epigastric, and cremasteric reflexes are absent. The right ankle-jerk is 3 plus, the left, 1 plus. Babinski and clonus are both present on the right side.

Abnormal Involuntary Movements.—There is a history of several epileptiform convulsions.

Muscle Strength.—Is much impaired in the right arm and right leg.

Muscle Status.—Practically unchanged in the right arm and leg. The *electrical reactions* are normal.

Abnormal Associated Movements.— The coughing sign of Huntington and the tibialis sign of Strümpell are both present.

NERVE STATUS.—No record.

GENERAL SENSORY.—There are no sensory changes.

CRANIAL NERVES.—The fundi are normal. There is a slight external strabismus of the left eye.

HISTORY AFTER THE ONSET.—This patient was at first practically wordless, being able to say only "Yes" or "No." Soon, thereafter, however, he began to use words, but spoke a jargon, and was only able to give his own name and that of his father and several of his relatives. He used "Yes" and "No" properly for assent and dissent, but aside from this, could not be said to have much spontaneous speech. He has always had the power of mimicry very highly developed, and can make himself understood by various means in this way. He learned to use his left hand very quickly in writing and does so fairly well. He is a great deal improved, particularly on the sensory side, being able to understand a great many more things that he sees and hears than he did at first.

DIFFERENTIAL DIAGNOSIS. — About one month ago the patient had a sudden attack of hematemesis and was taken to St. Catherine's Hospital in Brooklyn, where the *X*-ray diagnosis was said to be a possible cancer of the stomach. The surgical diagnosis, however, with which the patient was discharged without operation, was ulcer of the stomach. The hematemesis lasted several hours, was quite severe, but was finally controlled. The patient left the hospital a week later very much improved. He is now in a convalescent home, improved physically but not very much in regard to his speech.

LABORATORY TESTS. *Wassermann*.—Blood Wassermann has been negative on several occasions.

Blood Count is normal. Coagulation time of the blood is normal.

Urine.—Urine is negative.

X-ray.—*X*-ray has been negative and *X*-ray at St. Catherine's Hospital shows, according to their interpretation, a possible tumor of the pylorus.

Eye.—A report from the Eye Department by Dr. Holden gives the fundi as normal, and the external strabismus of the left eye as functional.

SUPPLEMENTARY PHYSICAL EXAMINATION. *Blood Pressure.*—The blood pressure is 120 systolic.

Pulse.—The pulse is 92, arhythmic, soft, and variable, on certain occasions going down as low as 68.

Glands.—No glands are palpable.

Lungs.—The lungs are normal.

Heart.—The heart shows a systolic murmur at the apex.

CASE III. R. B.; age 29; married; watchman by occupation; born in United States.

HISTORY.—*Present Illness, Beginning with Complaint.* The patient came to the clinic May 11, 1917, complaining of paralysis of the left side of the body, including face, arm, and leg; loss of memory, and as his wife expresses it, "foolishness." This condition has lasted three months and has remained stationary. The previous history shows that one and a half years before the onset of his present trouble he was hit by an automobile in the lower spine. The exact date was December 17, 1915. The automobile threw him to the ground and he was unconscious for two hours. He was taken to Bellevue Hospital where an *X*-ray examination showed nothing in the way of fracture or dislocation. He was discharged from the hospital with a diagnosis of contusion of the spine, and was returned to his home practically well, except for a soreness in the lower dorsal region when he bent forward. This pain in the back persisted for several months and finally disappeared. The patient's wife says that he has never been ill, and he says he has never suffered from any venereal infection. For two months before the actual onset of the present trouble, the patient complained of headaches and vomiting nearly every day, usually at night after supper. During this time, too, his general disposition seemed to undergo a change. He was usually irritable, restless, and gave way to explosive outbursts of temper. He did not complain at any time of dizziness, nor did he have perversions of other subjective changes of sensation in any of his limbs. There was no numbness or weakness and never any disturbance of speech during this prodromal period of two months before the onset of the aphasic condition.

The patient was in the habit of going to work at night as a watchman. At 1 o'clock his actual work was finished and from then until 7 o'clock in the morning it was his custom to sit on a chair or pass the time until morning as best he could. It is definitely known that until 1 o'clock on the morning of February 25, 1917, he had been in his usual health, in fact, it is known that for a few days before the onset he had had no headaches, no vomiting. Nothing is known about what occurred to him after 1 a. m. February 25, 1917, and he himself is not able to explain, excepting that he sat down to rest and to sleep until morning. He was not seen again and nothing is known about him until a chauffeur found him unconscious on the floor about 7 a. m. He was unconscious for an hour thereafter. When he regained consciousness he did not speak for more than a week. He was absolutely wordless. During the second week he began to call his wife by name (May) then his boy, Willie, and finally his sister, Hannah. He could say "yes" or "no," but did not always use the words properly, and he could ask for a drink. During the second week he was very ill-tempered. He remained in Bellevue Hospital for three weeks and during this period he could say no more than what has just been mentioned. He was five weeks in the City Hospital and here he improved physically and was able to get up and walk. Mentally he has remained practically the same until now. He has added but very little to his speech and the condition has continued practically the same since the onset.

Previous History.—It is well to note that the early history shows that this patient was born in New York City, began school at the age of six and graduated at the age of fourteen. He was bright and was promoted regularly, but was never very fond of attending school. After graduating, he went to La Salle Academy for one year and quit soon after his father died, in order to go to work. The only foreign language which he ever tried to study was French, for one year at La Salle Academy.

It cannot be said that he ever really knew very much French and he forgot what he did know long before the onset of his trouble. He was always considered a fairly good singer, but never had any musical education, either vocal or instrumental. He was always rather wild, preferred to dance and play pool, rather than study while he was at high school. He always preferred mechanical work to study of any kind. He never read anything except newspapers. Mechanically, he was never very skilled, and was only a laborer in the highways department of this city when the accident occurred. He, and all of his family are right-handed, including his hemiplegic sister and his mother. For a number of years he had been in the habit of drinking about one pint of beer a day.

Family History.—The family history in this case is very interesting. His father is dead; cause unknown. Mother died at the age of forty-three after an illness lasting only three days, from an attack of apoplexy. The onset in the mother's case was very sudden and she never regained consciousness, so that the supposition is that she had a cerebral hemorrhage. The rest of the family consists of two sisters. One of them is perfectly well physically and mentally; the other, aged thirty-two, had a right-sided apoplectic seizure two years ago, without a defect in speech of any kind whatsoever. She did not make a good recovery from this, although she improved somewhat until three weeks ago, when she had a second similar attack of right-sided hemiplegia.

The patient and his wife have had two children, both of them living and well. Wife never had any miscarriages.

VOLUNTARY MOTOR SYSTEM. *Attitudes.*—The posture is hypertonic.

Gait.—The gait is hemiplegic.

Coördination.—Good in parts not affected by paralysis.

Skilled Acts.—The following is a series of fifteen questions which have been put to this patient with a view of determining his aphasic status with a record of the answers as the patient made them:

Question 1.—Can the patient speak spontaneously in ordinary conversation? (What is your name?) (What is your trouble?)

Answer.—"Cold—none—storke—speech—I do not know." In other words, there is no spontaneous speech.

Question 2.—Can he enumerate, denominate, and name objects?

(*a*) Can you count your fingers aloud?

(*b*) Can you name the members of your family or

(*c*) The name of the month?

(*d*) Can you name these objects?)

Answer.—(*a*) Counts all fingers, then says "two." Asked "How many fingers on both hands?" says "four." (*b*) (Willie, Annie), says "Maggie," then "May," and then mentions the name of his niece, Genevieve. He was asked to limit his answer to the members of his immediate family. He, however, included his sister and niece (Hannah and Genevieve) and made a mistake at first in mentioning his wife's name, so that we may easily consider that this is a defective answer. (*c*) The answer to this question was August and the second time, September, which of course makes this an absolute failure. (*d*) When asked what an inkwell was, he responded "Ink." Pointing to a yellow sheet and asked for color and the name of the object, he answered "Paper." Pointing to a magazine his answer was "Book." Pointing to a blotter, his answer was "Boiler." Pointing to a rubber stopper in a bottle, his answer was "Celluloid." When asked to name the number of chairs in the room, he rose to close the door. When asked the color of his tie, he said "Blue," then, "I don't know." (The color of his tie was black.)

Question 3.—Can he speak from dictation?

Answer.—He cannot speak from dictation.

Question 4.—Can he read aloud?

Answer.—He cannot read aloud matter which is printed or written.

Question 5.—Can he read to himself and understand what he reads?

Answer.—When asked to read aloud from a newspaper article, he got up to look for a piece of chalk and finally asked for chalk by name. The only thing which he can read aloud is his own name, that of his wife and child, and very little else. He does not understand what he reads, even though the article is a simple one.

Question 6.—Can he recite what he reads? Can he say his prayers?

Answer.—He cannot recite his prayers with which he was previously very familiar. Cannot sing his favorite song, which was "River Shannon," cannot even hum it or whistle it tunefully. Tone and cadence are missing from the whistling.

Question 14.—Has he a concept of his speech?

Answer.—He appears to have no conception of his speech as tested by Lichtheim's test. He cannot tell how many syllables or letters there are, by any means of indication, in any words, not even his name. Occasionally he can tell the number of letters in his name and in the name of the president of the United States, but he is not always correct. He has at no time been able to tell us the number of syllables in any given word.

Question 15.—Does he misuse words? Is he ungrammatical out of proportion to his education? Does he perseverate? Does he speak a jargon?

Answer.—He misuses words. He is ungrammatical out of proportion to his education. He repeats fixed phrases and when he does speak or write, it is a jargon.

Reflexes.—Reflexes show that the left triceps is 3 plus and is much greater than the right in its reaction. This applies also to the left biceps. The left patellar is 4 plus with clonus. The left upper and lower abdominals are 0, those of the right 2 plus, and the left cremasteric is 1 minus, that of the right 2 plus. The left ankle-jerk is more active than the right. Babinski and Chaddock are present on the left and clonus is almost inexhaustible on the left side.

Abnormal Involuntary Movements.—There are no irregular involuntary acts except the usual ones associated with hemiplegic associative movements.

Muscle Strength.—The muscles in the left limbs are hypertonic. There are no tremors. Motor power in both limbs on the left side and in the face is much weaker than the right. The muscles in the left limbs are hypertonic. The electrical reactions show a hyperexcitability to both faradism and galvanism, but no change in the normal contraction formula.

General Sensory.—There are no subjective or objective sensory changes.

Cranial Nerves and Head.—As has been mentioned, the seventh, ninth, and possibly the twelfth are involved.

Mental Status.—The mental status shows some confusion. (*See* above answers.)

Laboratory Tests. *Wassermann*.—The Wassermann of blood and spinal fluid is negative, the patient having had numerous spinal fluid and blood Wassermann tests at Bellevue Hospital and the Vanderbilt Clinic.

Blood Count is normal. There is no change in the coagulation time of the blood.

Urine is negative.

X-ray.—*X*-rays of the skull and spinal column taken at Bellevue Hospital are negative.

Supplementary Physical Examination. *Pulse*.—The pulse is 70.

Temperature.—Normal.

Glands.—No glands are felt.

Heart.—Normal.

Lungs.—Normal.

Blood Pressure.—Blood pressure is 140 systolic and 90 diastolic.

Diagnosis.—The diagnosis of this case is, in my opinion, thrombosis, or a hemorrhage causing a left hemiplegia with a combined sensory and motor aphasia, resulting in paraphasia, paragraphia, paralexia, and apraxia. I am more inclined to the thrombus conception.

DISCUSSION

Dr. Goodhart.—The problem is a difficult one to take up for comparative analysis with a view to combating the theory that has held sway since the time of Broca. It seems to me the onset in this case was not definitely such as we see in any forms of the apoplectic type; there was evidence of intracranial disturbance some months previous. The aphasia itself is not a simple motor type and cannot be grouped as a simple sensory form; it is too complicated to be regarded as anything but a mixed form. It is in character essentially a motor affection, its basis a subcortical lesion. Though not purely a motor aphasia, it is the dominating feature. Stimuli coming from sensory centers do not reach the expressive centers. The intellectual defect here is an interesting phase and, taken as indicative of a generalized arterial disturbance, has a bearing upon the question of the localization of speech within the brain. The language defect in this case appears somewhat different from that which one sees ordinarily in any form of simple aphasia, motor, sensory, or mixed. Some responses were quite immediate and did not fit in with absolute deficiency to respond to other stimuli of identical character. I am impressed with the generalized pathological process in this case as revealed by the history and by the present examination. A partial thrombosis of the middle cerebral would be my suggestion, though intracranial pressure symptoms had preceded these definite manifestations. A generalized process, perhaps, with the etiological factor in the previous trauma, played its final stroke as an embolus, the result of cerebral arterial sclerosis, or only the result of slowing up of blood current, lack of arterial tone, not as the result of concussion. A simple "stroke" as the result of a distinctly circumscribed lesion, in a right-handed patient with a process in the right hemisphere might not have caused such profound effect upon speech, though of course such cases do occur. In line with my idea is the manifestly greater preservation of the power of motor expression by writing which is so striking in this patient. The motor technic with the left hand is relatively very good. It would seem that if speech were altogether a skilled act, writing, even with the right hand, in this case should be also very deficient unless the patient were ambidextrous.

Dr. Sharpe.—I do not see how the aphasic symptoms of this patient can be accounted for by Broca's theory. There have been several cases, though rare, in which the patient had left hemiplegia with aphasia. There is much evidence to show that the third left frontal convolution is the motor area of speech. Six months ago in operating on a patient I attempted to outline a tumor and passed across the third left frontal convolution and the patient was aphasic for a week after. This patient is a good example of the difficulty in relying on speech disturbance in diagnosing the location of a lesion. He would appear to me to be more a visual aphasic than an auditory one. As a matter of fact many of these symptoms depend on the individuality of the patient, one speaking and learning to speak by visual images, another by auditory. I am inclined to agree with Hughlings Jackson that speech is the function of the intelligence and anything that lowers the intelligence will interfere with the speech mechanism.

Dr. Hoyt.—The fact that so many of the family had the same trouble all within six years, seems significant. I do not believe it can be put on a hereditary basis, but there might be some etiological factor in the nature of an infection that affected all the family at the same time. There were no acute illnesses at the time of the hemiplegias, therefore the only etiological factor that could be a common source would be some form of food poisoning. The process was slow with gradual termination in hemiplegia.

Dr. Eleanor I. Keller.—I think this man's status might be partly accounted for by his social status, his lack of education, or misunderstanding of many of the questions put to him. I noticed he did things correctly when he was conscious of the meaning of what he was requested to do. The amount of deterioration would be computed by his real mental status before the onset of his malady. He may never have formed any logical forms of association. He has certainly fallen off from what was possibly his normal state, but I should not call all his present mental lack aphasic.

Dr. Frederick Tilney.—The collective evidence of the literature is remarkable, and is summarized thus by Moutier:[1] "Between the years 1861 and 1906, 304 cases with autopsy have been published bearing upon this subject. Of these cases 201 are not really of value, since in twenty-six cases the clinical or the pathological records were so incomplete as to give the reader no conviction as to the nature of the case, and in the remaining 175 cases the lesions were too vast for accurate localization.

"There remain, then, 108 cases in which the lesions were localized, and the records sufficient for purposes of argument. In nineteen only of these cases did the facts appear to support Broca's theory, and in eleven of these the lesion was confined to the subcortex; in eight only was the cortex of the left third frontal gyrus affected.

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[1] "Recent Work on Aphasia," *Brain*, 1908, Vol. XXXI, p. 533.

"The remaining eighty-four cases were entirely against the classical theory; in fifty-seven the left third frontal gyrus was intact, and motor aphasia was present. In twenty-seven cases this convolution was destroyed and there was no aphasia present, and in two of the latter cases the left third frontal gyrus was destroyed upon both sides."

According to Moutier, a careful examination of the nineteen apparently favorable cases shows that even these cases are the reverse of conclusive; in some there were multiple lesions, in one there was an extensive lesion of the right temporal lobe. Complete alexia was present, and there is no mention as to whether the patient was left-handed or not. He sums up the analysis with the words "there does not exist in medical literature one observation of Broca's aphasia in which a single lesion, strictly localized to the foot of the left third front gyrus, has been proved to exist upon autopsy."

SUMMARY OF DISCUSSION

Dr. Osnato.—Discussing this case from the standpoint of an explanatory diagnosis, I feel reasonably certain that we have to deal with either a thrombus or a hemorrhage in the middle cerebral distribution, despite the fact that prodromal symptoms existed for so long a period before the attack. This particular point, and particularly the headaches and vomiting with which this patient suffered for two or three months before the hemiplegia, has led Dr. Tilney to believe that this man's condition is due to a syphilitic process. However, I feel that I am standing on very firm ground when I hide behind the laboratory findings in this case, particularly the repeated negative blood and spinal fluid findings. Some of these Wassermanns were taken after provocative injections. Indeed, another point against the possibility of a syphilitic infection being the underlying cause of this man's aphasia and hemiplegia, is the fact that he did not improve despite two large provocative doses of salvarsan, repeated injections of mercury and also the administration of potassium iodide. I do not understand why we are not justified in believing that we have to deal in this case with a hereditarily insufficient vascular system. We, of course, know of numerous diseases in which degeneration, deformities, and arrest of development take place in various parts of the body. We are all acquainted with the familial types of myopathies and dystrophies and with the degeneration and arrest of development which occur in Marie's cerebellar ataxia and Friedreich's ataxia. Reasoning by analogy from this it seems possible that we have to deal in this case with a cerebral vascular system which is hereditarily defective. It may exist as a recessive trait in this family and may account for the early occurrence of cerebral hemorrhage in the mother who died in three days without regaining consciousness, and may also account for the hemiplegia in the sister and in this patient, both of whom are exceptionally young hemiplegics with no assignable cause for the trouble. Of course, this same argument may speak for hereditary syphilis as a cause in these cases, but I am more than anxious to call in again the lack of positive specific findings in the patient and his sister to controvert this theory. I do not see why it is not possible to hand down to posterity a weakened set of cerebral blood vessels, just as other defects are handed down. It is certainly strange that in a family of five, three should have hemiplegia.

In answer to Dr. Sharpe, I feel that we are still in need of absolute revision of our ideas of aphasia, as Marie pointed out ten years ago. Our conceptions of aphasia are not all clear; but it would seem that after a recital of Marie's statistics, quoted by Dr. Tilney, that Broca's area is not the keystone of our conception of the mechanism of speech. Dr. Tilney's conception that speech is simply the highest type of skilled act and that many parts of the brain, in association, take part in its production, is the correct one. It seems wise to divide fairly definitely the intellectual or internal part of speech from the effector or utterance part of speech. Of course, words are used in the service of speech by means of motor control, which is more or less located, possibly, in Broca's area, and this control is exercised over the muscles of the larynx, pharynx, and tongue to formulate and allow the utterance of words and sounds. There must be of necessity a very close association between the conceptions, which are internal speech, and their motor expression, which are words. It is probably true that Broca's area, or that region of the brain, has a great deal to do with the almost simultaneous discharge of motor stimuli which causes the production of sounds which we call words. There is scarcely any room for doubt that the schematic, sharply circumscribed areas which are known as centers for writing, reading, auditory word images, and visual word images, do not exist. The more probable explanation is that exact patterns for the production of speech are not followed continuously to particular portions of the brain, but that a great many parts of the cerebrum take a prominent place in the production of word images—both visual and auditory—perceptions, conceptions, and, finally, their utterance as speech.

This case seems to illustrate very forcibly the fact that these areas are not necessarily localized in the average normal individual on either one side of the brain or the other.

Dr. Tilney.—I consider that we are indebted to Dr. Osnato for this excellent presentation of his cases and also for his summing up of the subject in general.

A CASE PRESENTING THE THALAMIC SYNDROME

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Patient: S. S.; male; age 36.

HISTORY. *Present Illness, Beginning with Complaint.*—In July, 1917, examination showed that the patient was suffering from profound sensory loss on the right side of the body, accompanied by emotional states in which there was spontaneous outburst of laughter or weeping, independent of humor or distress. These phenomena dated back two years to the initial insult. There is now right facial flattening of the upper motor neuron type.

Previous History.—In July, 1915, upon retiring after an oppressively hot day the patient suffered from rather intense headache but slept fairly well and recalls having a most disturbing dream towards morning. Out of this dream he awoke in fear and from then on was aphasic with complete right hemiplegia. The content of this sleep experience is of interest. The patient dreamed that just outside of his window the Germans and Russians were waging a hand to hand combat, and that the Russians were being badly beaten. He determined immediately to go to the assistance of the latter, was intensely wrought up, and, it seems in a half waking state, made a violent effort to shout and to spring from bed. He realized for a moment that he was unable to speak and that his right side was powerless. The history obtainable then records that from this time on there was a period of three days of more or less complete unconsciousness, during which hemiplegia and hemianæsthesia of the right side were established and also an aphasia, probably of the motor type. The hemiplegia was almost fully recovered from within a short time. The aphasia returned a year later, but was only transitory. Later examination established the fact that concomitant with the motor paralysis there occurred profound sensory loss on the same side of the body, accompanied by emotional states in which there were spontaneous outbursts of laughter or weeping, independent of humor or distress.

Family History.—Other than diabetes in the direct line no ancestral taint appears. Personal history gives prominence only to years of excessive cigarette smoking and alcohol moderately indulged in. In so far as careful history and repeated examinations can be relied upon, syphilis could be excluded. The patient has two healthy children, and his wife has had no miscarriages.

VOLUNTARY MOTOR SYSTEM. *Attitudes.*—None recorded.

Gait.—Though only slightly affected the gait is of the hemiplegic type.

Coördination.—Equilibratory tests are well performed. Non-equilibratory tests establish the presence of ataxia of the right upper and to a lesser degree of the right lower extremity. There is adiadokokinesis of the former. (See under sensory examination.)

Skilled Acts.—There is no apraxia and no dyspraxia. Speech is of the explosive type, at times suggestive of pseudobulbar palsy; furthermore, it shows an ataxic element. The voice is monotonous in character. There is no aphasia. The handwriting is affected only by the abnormal movement of the upper extremity, the patient being right-handed.

[1]*Deep Reflexes.*—(1 signifies slight, 2 active, 3 very active, 4 with clonus; 0 means absent).

	Right		*Left*
Patellar	3		2
Biceps	2	=	2
Triceps	2	=	2
Achilles	4		2

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[1] These numbers conform to a scale regularly used in the Neurological Department to give uniformity in estimating degrees of reflex activity.

	Right		Left
Jaw-jerk	R	=	L
Pectoral	R	=	L
Wrist	R	=	L
Radial	R	=	L
Ulnar	R	=	L

Superficial Reflexes.—

	Right		Left
Ciliospinal	R	=	L
Supra umbilical	0		2
Supra-public	0		2
Abdominals	0		2
Cremasteric	1	=	1

Plantar and Its Modifications

	Right	Left
Plantar flexion	0	2
Babinski	0	0
Chaddock	0	0
Oppenheim	0	0
Gordon	0	0
Schaefer	0	0

Abnormal Involuntary Movements.—A choreiform, athetotic movement of the right upper extremity is present, the hand and fingers being especially involved; the abnormal movement is much exaggerated during emotion, pleasurable or otherwise; in lesser degree the right leg is similarly affected.

Muscle Strength.—There is slight but manifest relative weakness of the right upper and lower extremity.

Muscle Status.—There is slight hypertonus of the muscles of the right arm and leg. Electrical reactions show no change from the normal formulæ.

Abnormal Associated Movements.—None of the 'small signs' of hemiplegia are present.

NERVE STATUS.—Not recorded.

GENERAL SENSORY. *Touch.*—(*a*) Objective.—Over the entire right half of the surface of the body, not exactly corresponding, however, to the mid line, stimuli are not felt. The anæsthesia includes the sensory distribution of the right trigeminal nerve, the cornea, and mucous membranes included. (*b*) Subjective.—No dysæsthesia.

Pain.—Rather severe pain indefinitely described by the patient but said to have been of a 'peculiar' character, and confined to the entire right upper extremity continued for some months from the beginning of the illness. Circumstances of aggravation or alleviation did not appear to exist. The pains were not of nerve or root zone distribution.

Paræsthesia.—Stroking the anæsthetic parts, especially the arm and abdomen, with a rough surface, at times even with cotton wool, provokes sensations of a disagreeable character. The patient describes them as "unangenehm" (unpleasant). It is of interest to note that this phenomenon is not constant, but at times absolutely positive.

Temperature.—Stimuli of intense degree show an existing analgesia and thermal anæsthesia over the same area as for tactile impressions. Applications of heat and cold cause simply disagreeable sensations.

Vibratory.—Tuning fork stimuli are not appreciated upon the osseous parts of the right half of the body.

Pressure and muscle-tendon sensibility are both defective on the right side of the body. There is astereognosis and barognosis of the right upper and lower extremities.

CRANIAL NERVES AND HEAD.—The first, second, third, and fourth nerves show no defect. The sensory changes in the distribution of the fifth have been mentioned. The seventh shows paresis of the supranuclear type. A difference in voluntary and emotional facial activity cannot be definitely made out though at times the paresis seems to disappear during strong emotional reaction. Articulation of labials is defective. There are no vasomotor changes. In the innervation of the ninth, tenth, and twelfth nerves, there are phonation and voice quality changes.

MENTAL STATUS.—There is no evidence of abnormality. The emotional reactions are rather of the spontaneous, automatic type, not dependent upon psychic disturbances.

SYSTEMIC CONDITION. *Tegumentary.*—Tegumentary status and mensuration of corresponding parts of the body give no pathological findings.

Glandular, Cardio-vascular, Respiratory, and Gastro-intestinal systems show no abnormalities. Blood pressure, systolic and diastolic, is equal on both sides and within normal limits.

Genito-urinary.—Rectal, vesical, and sexual control is unimpaired.

Laboratory Tests. *Wassermann.*—Negative of the blood.

Lumbar puncture.—Negative.

Differential Diagnosis.—The following are arranged in the order of their seeming importance. (1) Sensory changes especially affecting deep sensibility confined to right side of body. (2) Hemiplegia with almost complete recovery. (3) Paræsthesia of a special character. (4) A definite type of emotionalism. (5) Hemiataxia. (6) Hemichoreoathetosis. (7) Spontaneous pains. (8) Onset in dream state.

The first seven of the above symptoms point with reasonable certainty to a lesion of the optic thalamus.

The etiological factor is not so clear. A vascular lesion is most likely, though the age of the patient and the condition of the vascular system, including the heart, give this no substantiation. Specific infection of remote or direct origin is always a possibility though its likelihood in our case seems to be ruled out.

The organic nature of the sensory disturbances appears established. The area of abnormal sensation is not exactly demarcated by the mid line of the body anteriorly and posteriorly; the mucous membranes and cornea are included in the affected parts. All of the qualities of sensation are involved; the elements of postural sense, as is not infrequent in thalamic cases, are profoundly disturbed. The organic nature of the hemiplegia is shown as the typically transitory type; by the mild degree of paresis without contractures; by the behavior of the deep and superficial reflexes; the facial paresis of the upper neuron type, the superior facial muscles not being involved.

The very marked degree of sensory loss, as seen in this case, though not usual in the thalamic syndrome, may well be expected to occasionally occur in certain lesions of this great somæsthetic relay with its centers of extensive association. Much of our knowledge of these thalamocortical relations is, of course, still speculative. However, it is probable that the removal of cortical inhibition from the thalamus, giving the latter its uncontrolled over-activity upon slight stimulus, accounts for the seeming emotional outbursts, and perhaps also for the typical affect reaction, when slight sensory stimuli are applied to the anæsthetic parts.

The relationship of the patient's dream to the initial insult is of especial interest. It is, of course, easily conceivable that the dream experience was given its impressive content by an irritative lesion, first in the thalamus, then along thalamocortical fibers; or again, circulatory variation in an active brain state in dream might be a cause of sudden increase of blood pressure acting upon sclerotic vessels.

The association, however, may be more subtle and rather invites speculation into the psychopathology of the subject. I recall among some experiences of this character, one case which especially impressed me. A female patient at the Montefiore Home and Hospital with a fully developed Parkinson syndrome, related that the first symptom noticed by her was a 'shaking' of one upper extremity. This appeared upon awakening in the morning from a disturbing dream experience in which the patient was bitten by a dog in the hand affected.

One might easily go far afield in speculation but the subject invites further study; any observation that touches that great borderland between the organic and the functional in neuropathology is worthy of record. The case above described presents the salient features of the classical thalamic syndrome.

DESCRIPTION OF A SUMMARY AND DIFFERENTIAL DIAGNOSIS BLANK

By OLIVER S. STRONG, Ph.D.
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It has been considered worth while to describe here a blank used in the Vanderbilt Clinic in making summaries and differential diagnoses of neurological cases. While the method it illustrates makes no especial claim to originality, yet it is so easy of adaptation to other systems of examination that it may prove useful elsewhere.

The blank is printed on one side of a double sheet twice the size of the pages of the regular examination blanks used in the clinic. On the left of the first, or left-hand page, is printed in a single column the various heads or divisions of the long blank used in making more extensive neurological examinations. The rest of the whole sheet to the right is lined, each head having one line. In addition to these horizontal lines, the second, or right-hand page, facing the first, is divided into nine vertical columns (the number is not important).

The first step in using the blank is to enter a summary of the findings under each head, a simple procedure inasmuch as the original examination was made under the same heads and, therefore, no regrouping of the findings is necessary. Usually the single line on the first page assigned to each head is sufficient, but in the case of an anamnesis which cannot be condensed so much the blank reverse side of one of the pages may be used.

Having entered the summary of the case, the next step is to enter at the heads of the vertical columns on the second page the various possible diagnoses, one diagnosis to each vertical column.

The next step is to give each finding under each head a number in each vertical column, this number representing its probability of occurrence percentage under the diagnosis at the head of the column.

After this has been done, the numbers in each column are added, the totals indicating the total probability of each diagnosis on a percentage basis.

Such a use of numbers may appear to give a false precision to the probabilities indicated, and in a sense this is true; nevertheless, numbers enable the clinician to convey his judgment more concretely, precisely, and briefly than any other method of designation. It may seem, for example, in a given case rather absurd to estimate the probability of a certain clinical finding occurring in one diagnosis at 8 and in another at 7, with practical certainty put at 10; yet such numbers convey clearly and concisely the idea that the clinician considers the first (8) as quite or very probable and the second (7) as probable but somewhat less so.

A possible source of error might arise in not recognizing the fact that some findings (*e.g.*, Argyll-Robertson pupil) have much more diagnostic importance than others (*e.g.*, pain or some slight sensory change). This error can be avoided by 'weighting' certain more important symptoms by adding a certain amount to their number or multiplying it by some low coefficient.

Another advantage in the method lies in the mere writing down all the possible diagnoses. This, like writing the numbers, gives the whole procedure greater concreteness and definiteness, and possibilities are dealt with which might otherwise escape consideration.

The blank under consideration has also other uses: it gives a definite order to be followed in the presentation of a case at a clinical staff conference, and those present who are provided with unfilled blanks can easily follow

TITLE Initials of Patient	AUTHOR Case No	INTRODUCTORY PARAGRAPH

Age Sex S. M. W. Race Nationality Occupation........................

A. History 1. Present illness, beginning with complaint........................
2. Previous history........................
3. Family history........................

B. Voluntary motor system. 1. Attitudes........................
2. Gait........................
3. Coördination........................
4. Skilled acts........................
5. Reflexes (a) deep........................
(b) Superficial........................
6. Abnormal involuntary movements........................
7. Muscle strength........................
8. Muscle status........................
9. Abnormal associated movements........................
Summary and interpretation of B........................

C. Nerve status........................

D. General sensory. 1. Touch........................
2. Pain........................
3. Temperature........................
4. Vibratory........................
5. Pressure........................
6. Muscle-tendon........................
7. Stereognosis........................
8. Barognosis........................
Summary and interpretation of D........................

E. Cranial nerves and Head. 1. Olfactory and Nose........................
2. Optic and Oculomotor Apparatus........................
3. Trigeminal and Mouth........................
4. Facial and Face........................
5. Acoustic and Ear. (a) Cochlear........................
(b) Vestibular........................
(c) Ear........................
6. Glossopharyngeus, Vagus, Pharynx and Larynx........................
7. Spinal accessory........................
8. Hypoglossus and Tongue........................
Summary and interpretation of E........................

F. Mental status........................

G. Cranial morphology........................

H. Systemic. 1. Tegumentary........................
2. Glandular........................
3. Cardio-vascular........................
4. Respiratory........................
5. Gastro-intestinal........................
6. Genito-urinary........................
7. Skeletal........................
Summary and interpretation of H........................

I. Laboratory tests........................
1. Wassermann........................
2. Lumbar puncture........................
3. Blood count........................
4. Urine........................
5. X-ray........................

J. Differential diagnosis........................

Reproduction of the Summary Blank. (Original size of sheet upon which this blank and its continuation, the Diagnosis Blank, appear is 11 inches high and 17 inches wide.)

For differential diagnosis: Enter possible diagnoses at heads of columns. Indicate by numbers the probability of occurrence of each clinical finding in each diagnosis.

Reproduction of the Differential Diagnosis Blank.

and note down in the proper places the findings as they are mentioned by the speaker.

Similarly the blank also provides a definite order to be followed in preparing cases for publication.

A final advantage of a blank with a definite list covering all the points which may be investigated in a neurological clinical examination is that it makes easy the noting of *negative* findings, a point of great importance from the point of view of a record. This common failing of case records has been mentioned elsewhere. If no mention is made of, *e.g.*, sensory findings, it is impossible to tell whether the sensory findings were negative or whether sensation was not investigated at all. This point is of equal importance in recording the original observations, *e.g.*, in the anamnesis of a case the remark that there was no past history of other diseases does not have much value unless it is known what diseases were inquired about. In the same way the statement that muscle strength was normal is of doubtful value unless the original record makes it clear what muscular movements were investigated, whether simply hand grips or a systematic testing of the various movements of all the members. A long blank is in course of preparation which will give such a record of every observation actually made whether the findings be positive or negative.

The general plan of assigning percentage numbers to each finding under various diagnoses was originated and successfully employed by Dr. Tilney on a blank with a fixed grouping of findings irrespective of the examination blank. The above blank combines this with a convenient summary blank so that no regrouping of the findings is necessary.

The actual headings used in the Vanderbilt Clinic blank are given on pp. 26-27. It will be noticed that the minor subheadings are usually omitted but it may be well to add the following: A2. Previous history includes (*a*) Character of birth; (*b*) Status at birth; (*c*) Development; (*d*) Diseases, Injuries, etc.; (*e*) Previous and Present Habits and General Physiological Status. B8. Muscle status includes (*a*) Volume; (*b*) Contour; (*c*) Consistency; (*d*) Tone; (*e*) Electrical Reactions; (*f*) Irritability; (*g*) Myoidema. C. Nerve status includes (1) Volume; (2) Consistency; (3) Irritability. Electrical tests are not listed under I, Laboratory tests, because they are already listed under B8 (*e*).

This is, as above stated, the system under which case reports from the Vanderbilt Clinic will be published in the Bulletin.

A CASE OF SYRINGOMYELIA WITH A DIFFERENTIAL DIAGNOSIS

By OLIVER S. STRONG, Ph.D.
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NEW YORK CITY

This case is presented in order to illustrate the preceding method of differential diagnosis. The history is as follows:

Patient: F. G.; case 8721; female; age 26.

History.—*Present Illness, Beginning with Complaint.*—On July 16, 1917, the patient said the right hand was flatter, and she felt fatigue in the knees if she walked six blocks. Other symptoms were the same as on the first visit.

Previous History.—On October 25, 1916, the patient came to the clinic with the complaint that she could not use her left arm. The condition had been gradual and progressive since three years ago when she noticed a weakness of the left thumb muscles, gradually extending to hand and up arm and shoulder. There were burning pains in left shoulder and arm (and occasionally in right shoulder). The left hand felt numb all the time. In the past year she noticed that the right hand had been getting weak and that the muscles were wasting.

Family History.—The family history is negative.

Voluntary Motor System. *Attitudes.*—Normal except possibly fingers and wrist (coöperation poor). On August 1 there seemed to be possibly some diminution in R. fingers, L. normal (?). Wrist?

Gait. — Normal. Some unsteadiness in Fraenkel's test rigidly carried out.

Coördination.—R. arm and finger ataxia in finger-to-nose test (left arm too weak to test well). Heel-to-knee test normal. Station normal. No Romberg.

Skilled Acts.—Nothing noted.

Reflexes.—(0 = absent, 1 = slight, 2 = active, 3 = very active), (*a*) Deep.

	Right		*Left*		*Right*		*Left*
Jaw-jerk	2	=	2	Patellar	3+	=	3+
Triceps	1?		0?	Suprapatellar	3+	=	3+
Biceps	3	<	3	Achilles	3	=	3
Wrist	1?	=	1?	Dorso-cuboidal	2	=	2
Radial	0		0	Periosteal	2	>	2
Ulnar	0		0	Crossed periosteal R to L?			

(*b*) Clonus.

	Right	*Left*		*Right*	*Left*
Ankle	0	0	Patella	?	?

(*c*) Superficial.

	Right		*Left*
Abdominals	0		0
Plantar	1	=	1 (sluggish)
Babinski	0		0

Abnormal Involuntary Movements.—Twitching of muscles of hands, especially of fingers, and also of wrist and of biceps of left arm.

Muscle Strength.—(*a*) Neck.—Turning head to R. and L., about equal, moderate weakness; bending to R. and L., weak; backward and forward, some weakness, especially forward. (*b*) Shoulders.—Elevation normal, retraction apparently normal (diminished?). (*c*) Upper arm (shoulder joint).—Abduction, cannot raise L. arm above horizontal; R. arm weak but can be raised; adduction at side fair, R. and L.; adduction in front (pectorals), weak. (*d*) Forearm (elbow joint).—Flexion weak, R. and L.; cannot raise left hand or forearm. Extension better than flexion but diminished R. and L. Some contraction on left. Pronation weak, R. and L. Supination weaker than pronation. (*e*) Hand (wrist joint).—Dorsal flexion, R. weak, L. limited in extent but some strength in holding it in flexed position. Ventral (volar) flexion better than dorsal flexion but diminished

R. and L. In all these tests the right side showed greater strength. (*f*) Fingers.—Position noted under *Muscle Status*. Flexion weak at knuckles, better in terminal phalanges. R. and L., R. > L. (cannot close left fist completely), flexion of little finger very weak. Extension better; not so good in terminal phalanges of fourth and fifth fingers (R. and L., R. > L.). Adduction and abduction limited and weak, about nil in left. Thumb adduction very weak, R. and L. (R. > L.); abduction better but weak, extension R. nearly normal, L. weak; flexion weaker than extension. (*g*) Trunk.—Extension dorsal (arching back), good. Can rise to sitting position and hold it well. Rotation of trunk diminished? (*h*) Upper leg (hip).—Adduction and abduction normal (latter?). Flexion fair? (R. slightly > L.?) Extension good (R. slightly > L.?). (*i*) Lower leg (knee).—Flexion R. and L., fair at first but apparently cannot hold it long (R. slightly > L.). Extension R. and L., good. (*j*) Feet.—Normal except dorsal flexion, R. possibly slightly weaker than L. Rotation in and out good. (*k*) Toes.—Flexion and extension normal.

SUMMARY. Paresis involving segments C.$_1$—Th.$_1$, R. and L., but most marked in deltoids, pectorals, biceps, supinators, interossei, adductor pollicis, and abductor digiti, represented in segments C.$_5$—Th.$_1$

Muscle Status.—Diminution in volume of right and left thenar and hypothenar eminences, more on the left, also of palm and back of hands, especially the left, and of the adductors of thumbs. Tendency to flex the terminal phalanges and hyperextend at the knuckles (claw hand), more pronounced on the left. Atrophy of biceps, especially the left, L. deltoid < R., flattening over scapulæ (subscapulæ), especially the left. These tests show muscular atrophies involving segments C.$_5$—Th.$_1$. In general, hypotonus is noted in muscles affected. Contraction of extensor (triceps) of left lower arm, of extensors of proximal phalanges, and flexors of terminal phalanges. (*a*) Electrical reactions (Dr. Blakeslee).—Faradism fair on R. in biceps, triceps, flexor group of hand, and extensor group of hand; diminished in above on L. except flexor group of hand. In interossei, flexor pollicis brevis, extensor pollicis, faradism is diminished both R. and L., but more on L. Galvanism is fair in all the above on R. except the extensor pollicis which is diminished and is diminished in all above on L. The adductor pollicis shows R. D., R. and L., more marked on L. Muscles of left upper arm and forearm: extensor surface show fatigue upon slight electrical stimulation with recovery in about five seconds.

Abnormal Associated Movements. — No record.

NERVE STATUS.—No record.

GENERAL SENSORY. *Touch.*—Slight hypæsthesia of left face (?), ulnar side of left palm, dorsum of two distal phalanges of R. fingers and distal phalanges of R. thumb, whole of R. palmar surface (? Aug. 1, not confirmed) apparently R. hand < L. Also right side of neck in part of C.$_3$ — C.$_4$ region (constant and more marked). Some evidence of a slight blunting over the whole upper trunk and arms when compared with legs. (*a*) Discrimination.—Thumbs, forefingers 7 mm., other fingers 4 mm.+ except R. little finger which is hypæsthetic and difficult to test. (*b*) Dysæsthesia. — Burning sensations (mentioned under *Tegumentary*) of left upper arm and shoulder, and extensor surface of lower arm, also a feeling as though sand or dirt were on volar surface of R. hand and fingers.

Pain.—Analgesia or deep hypalgesia in R. face (less near mouth?), back of head, neck, R. and L. arms and hands (less in fingers, tips of left fingers normal or hyperalgesic) and trunk to about Th.$_{10}$. (Sensibility begins to return rather higher than navel line and increases to about Th.$_{12}$; same on back.) Legs normal, except probably some diminution on left lower leg.

Temperature.—Practically same as pain. L. face (?), mouth (Oct., 1916), inside cheek R. < L.; tongue warm, R. = L., cold, R. < L.; inside upper lip warm, R. = L., cold, R. = L.; hard palate warm, R. < L., cold, R. < L. On back, cold sensibility begins Th.$_4$ or $_5$ and increases to about Th.$_{10}$; on front, begins at breast and at navel line is normal (or hypersensitive?). The abdomen is normal or hyperæsthetic and the thigh and also possibly the calf are hyperæsthetic to cold.

Warmth and cold are not felt as well on the front of the legs as on the back. R. > L. ? especially to warmth.

Vibratory.—Absent in R. elbow, shin (?), ankles, and big toe; present (diminished?) on left.

Pressure.—Probably normal.

SUMMARY. *Pain.*—Temperature, dysæsthesia in portion of L. C.$_4$ — C.$_5$ and L. C.$_8$ Th.$_1$. Touch, dysæsthesia in R. C.$_7$—Th.$_1$. Touch diminution in portion of R. C.$_3$ — C.$_4$, R. C.$_7$? — Th.$_1$, also (?) L. C.$_6$? — Th.$_1$. Discrimination also diminished. Pain diminished or lost, R. and L. C.$_1$ — Th.$_{10}$, except portion of left C.$_6$ — Th.$_1$ and R. V. (less near mouth?). Temperature nearly the same as pain, including R. inside mouth (less near the orifice?). Hyperæsthesia to cold, L.$_2$—$_3$+? R. and L. vibratory diminished or absent on right (arm and leg). Pressure probably normal.

CRANIAL NERVES. *Olfactory and Nose.*—No record.

Optic and Oculomotor Apparatus.—(Dr. Holden, Nov. 2, 1916.) Corneal sensibility reduced on R. Pupils slightly irregular. Light reaction good. Convergence reaction good. No diplopia. Rotatory nystagmus, R. > L. (more marked, coarse, looking to R.) (slow movement clockwise to observer).

	Syringomyelia	Amyotrophic Lateral Sclerosis	Multiple Sclerosis	Tabes	Neoplasm of Cord	Neuritis	Cerebrospinal Lues Meningomyelitis	Muscular Spinal Atrophy	Muscular Dystrophy
History	10	10	8	10	8	5	8	10	10
Inspection	10	9	0	2	7	5	9	9	0
Coordination	8	0	7	6	8	6	8	0	0
Gait	0	0	0	0	0	0	0	0	0
Skilled Acts	0	0	0	0	0	0	0	0	0
Reflexes	10	10	0	2	7	5	9	10	0
Muscle Strength	10	0	0	2	7	5	9	10	0
General Sensory	10	0	3	3	6	5	7	0	0
Nystagmus	7	0	10	1	0	0	1	0	0
Fundi Normal	10	10	5	5	10	10	7	10	10
	75%	39%	33%	31%	53%	41%	58%	49%	20%

The rating given to each sign or symptom is in proportion to the probability of its incidence in the disease in whose column it is tabulated.

Vision, 20/50 each. Hyperopia. Fields complete. Fundi normal.

Trigeminal and Mouth.—See above.

Facial and Face.—Facial movements, normal.

Acoustic and Ear.—Cold water Barany, patient recumbent; normal as to nystagmus (direction) and pass pointing.

Glossopharyngeus, etc.—Uvula, normal.

Spinal Accessory.—See above.

Hypoglossus and Tongue.—Taste anterior tongue, bitter tasted R. and L., sweet on neither. Tongue movements, normal.

MENTAL STATUS.—No record.

CRANIAL MORPHOLOGY.—No record.

SYSTEMIC. *Tegumentary.*—Pigmented area on left and right lower cheeks, less on right. Redness and heat over left shoulder, deltoid, and outer surface on left arm. Hands are cold, but patient says they feel warm. Cheeks flushed and hot. July 20, 1917: Patient sunburned her arms five days ago, says they were equally exposed, but the left was more sunburned, was swollen, as was the hand. July 24: Swelling partly subsided, marks of burns on hands and fingers.

Glandular.—No record.

Cardio-vascular.—Some evidence of vasomotor disturbance.

LABORATORY TESTS.—No record.

THE HISTORY OF A CASE OF ACROMEGALY WITH GIGANTISM IN THE FAMILY

An account of this case is presented in the patient's own words, in this particular instance, because of the unusually clear and pertinent character of the presentation. It is illustrative of the unusual practical value which may attach to a sympathetic perusal of an intelligent patient's impressions of the development of his own trouble.

[It occurs to the Editors that much information of great value could be conserved and reliable data adduced to help in the elucidation of difficult clinical problems, if patients were encouraged to manifest the same degree of healthy introspective and retrospective interest in the development of their conditions as was shown by this individual. To obtain this result, of course, the individual patient's capabilities must be considered, for here we were unusually fortunate in having an intelligent individual coöperating with us. Nevertheless, even with less brilliant patients, it should be possible by following this same plan to obtain reliable information. This line of investigation is too often apt to be disregarded, and we quite frequently do not pay enough attention to our patient's account of his difficulties.—EDITORS.]

GENTLEMEN:

I thank Dr. Tilney for his very gracious words of introduction. He made reference to the fact that I had been operated on by Dr. Harvey Cushing as being a distinction. In my particular case the operation came near being an 'extinction' instead of a 'distinction'. However, I am quite sure the fault was not Dr. Cushing's. I feel that I am discussing a subject about which I know almost nothing; but, of course, it is at the same time one in which I am more or less directly interested from a personal standpoint.

My first acquaintance with the word 'acromegaly' came as a result of an application on my part for life insurance, which application was refused by the company "because the applicant showed acromegaly on examination." A few weeks ago I wrote Dr. Cushing because I hoped to consult him at Boston before the opening of the summer session, as I am tempted at times to think my bones are still growing. A reply from Dr. Cushing's secretary stated the doctor was in France and would probably remain there till the end of the war. Not knowing exactly how long this would be I consulted Dr. McCastline at Columbia and thus was put into touch with Dr. Tilney, at whose request I am here this afternoon. I do not know exactly what he wants me to say as he gave me very little coaching (but since he brought me here my speech is, I suppose, like the tail of a dog, 'bound to a cur'. I assure you, however, that it shall not be like the tail of a cat, 'fur to the end').

I am not twenty-eight years old although I probably look to be several years older than that. In my early boyhood the bones over my eyes began to become very prominent and I remember being told by my mother to try to exert pressure on the bone by rubbing it with my hands. Of course the rubbing did not stop the growing. In 1909 my vision became greatly impaired, and I was examined by a specialist in Louisville, Ky., who said that "the choroid of the eye was inflamed." He prescribed glasses for me which, incidentally, never did my vision any good and also prescribed for me an alterative, which I did not like and from which I seemed to receive very little, if any, benefit. About that time I met an aunt on the street and passed her without recognizing her. As my vision continued to be very bad and the family thought I would almost surely become blind, they took me to a surgeon who circumcised me and removed hemorrhoids. My eyes soon got better and I began teaching again. About two years later, being then located in Oklahoma, I consulted Dr. Moulton of Ft. Smith, Ark., about my eyes. He said that the choroid may have been inflamed at the time of the previous examination, but at the time he made an examination there were no spots on the choroid, so he thought the trouble was with the optic nerve. I have no technical knowledge of the subject, but I am under the impression that the latter diagnosis must have been the correct one, as I suppose the enlargement of the pituitary would cause pressure on the optic nerve. My eyes are doing quite well now, although my left eye is practically blind. I have practically no field of vision on the left side of that eye and frequently have collisions with persons approaching me on my left side. The only other person I have seen whom I know to have acromegaly was totally blind.

Naturally after I had been refused insurance, I was quite eager to learn about acromegaly. The physicians around my home, a small town, did not know very much about the trouble, but in the summer of 1913, I attended the University of Chicago. One day while I was eating at the University Commons a young man introduced himself to me and asked whether I had ever spoken to anybody about my acromegaly. He had diagnosed the case from my facial appearance. He told me that Dr. Cushing, then of Baltimore, was the leading authority in the country on the trouble, and that next to him, Dr. Dean Lewis of Chicago had probably done the most investigating along that line. When I saw Dr. Lewis he had me go twice to the Presbyterian Hospital in order that he might conduct tests to find my sugar tolerance. Ordinarily, I believe fifty grams of dextrose will cause sugar to pass through the kidneys, but he gave me 150 grams without any signs of sugar in the urine. I have long been very fond of sweets and at times have left a table spread with meat and vegetables and gone to get candy in order to feel as if I had dined. Dr. Lewis told me that I could evidently handle candy better than most people and if I wanted to eat it, it would be all right to do so. About this time I would frequently have headaches in the occipital region and eating would seem to relieve the pain. I suppose putting food in the stomach called the blood from the head and relieved the pressure.

In August, 1914, I went to see Dr. Cushing at Boston, as he had by this time become connected with Harvard Medical School. I went hoping that an operation would not be necessary, but the doctor seemed to think that was the thing to do. So he made an entrance into the head between my upper teeth and lip and behind the nose and I suppose removed part or all of the pituitary body. In my hurry to get back to Oklahoma by the early part of September I did not stay at the hospital as long as I probably should have done. After reaching my home in Kentucky I became quite sick and during the next two months there followed a period of high blood pressure and delirium. That period is almost blank in my mind till this day, but my folks told me that I imagined myself to be president of the United States. I suppose infection was the cause of the trouble.

Since the operation my general constitutional condition has been better. My headaches have been much less frequent, and, although I still like sweets, I can probably do without them more easily than formerly. I think, however, that my bones are still growing. I have only about two or four teeth that occlude and they do so only with a glancing movement. Recently my step-father, a dentist, made an upper set of teeth to fit over my natural ones and they help me greatly in chewing.

I believe the generally accepted theory is that if the abnormal growth starts in infancy it causes gigantism and if it starts about the age of puberty it causes acromegaly. An interesting point in this connection is that an uncle of my father, Capt. Bates, was over seven feet tall. Gould and Pyle's 'Anomalies and Curiosities of Medicine,' has this to say about him: "On June 17, 1871, there were married at Saint Martins-in-the-Field in London, Captain Martin Van Buren Bates of Kentucky, and Miss Anna Swann of Nova Scotia, two celebrated exhibitionists, both of whom were over seven feet. Captain Bates, familiarly known as the 'Kentucky Giant', years ago was a familiar figure in many Northern cities, where he exhibited himself in company with his wife, the combined height of the two being greater than that of any couple known to history. Captain Bates was born in Whitesburg, Letcher County, Kentucky, on November 9, 1845. He enlisted in the Southern army in 1861, and although only sixteen years old was admitted to the service because of his size. At the close of the war Capt. Bates had attained his great height of 7 feet 2½ inches. His body was well proportioned and his weight increased until it reached 450 pounds. He traveled as a curiosity from 1866 to 1880, being connected with various organizations. He visited nearly all the large cities and towns in the United States, Canada, Great Britain, France, Germany, Spain, Switzerland, Austria, and Russia. While in England in 1871, the Captain met Miss Anna H. Swann, known as the 'Nova Scotia Giant', who was two years the junior of her giant lover. Miss Swann was justly proud of her height, 7 feet 5½ inches. The two were married soon afterward. Their combined height of 14 feet 8 inches marked them as the tallest married couple known to mankind. Beach describes the birth of a young giant weighing 23¾ pounds; its mother was Mrs. Bates, formerly Anna Swann, the giant who married Capt. Bates. Labor was rather slow, but she was successfully delivered of a healthy child weighing 23¾ pounds and 30 inches long." I think, however, that the child referred to died in infancy. The New International Encyclopedia quotes Stern as saying that forty per cent of giants have acromegaly and twenty per cent of acromegalics are six feet when the symptoms begin. My height is only a fraction over 5 feet 9 inches.

Morgan T. Craft

London, Ky.

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A CASE OF CEREBRAL NEOPLASM, PROVING TO BE CARCINOMA OF NASOPHARYNGEAL ORIGIN*

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In presenting the following case I must ask the indulgence of the Conference for not being able to establish a definite diagnosis, but it seemed to me of such extraordinary interest that I have asked the patient to come here this afternoon, preparatory to sending him to the Presbyterian Hospital for further observation and possibly surgical treatment. As the patient is suffering acutely and getting rapidly worse, I have considered it advisable to present him before obtaining the opinion of the throat and ear men or subjecting him to *x*-ray and transillumination tests.

Patient: A. M.; age 31; male; tailor by occupation.

History.—The patient came to the clinic a week ago with the following complaints:

1. "Itching of the right side of the tongue."
2. A sensation of coolness in the right side of the face.
3. Pain in the temporal region and forehead of the right side.
4. A sensation of being pulled outward in the right side of the lower lip.
5. The right eye is turned in.

The onset was gradual and the course has been progressive. Lues and gonorrhea were denied. He had never had any serious illness up to the onset of his present trouble. The family history was entirely negative.

Present Illness.—On August 15, 1917, he was operated upon for a swollen gland on the right side of the neck. The glandular tissue was removed and he was told that it was tuberculous in nature. He made an uneventful recovery.

A week after the operation he felt pain in the temporal region on the right side. Then he felt 'itching' sensations in the right side of his tongue which was soon followed by a sensation of coolness of the right side of his face. All of these manifestations appeared within a period of two weeks. In the middle of October he suddenly observed that he had double vision, and that the right eye had turned in. Attempts to use his eyes made him dizzy. The first three symptoms have progressed steadily since the onset, the last one remaining the same. His suffering has been so great that he has been unable to sleep well because of distressing sensations in the tongue. (While he is telling me of his experience, he is much distressed by what he characterizes as the biting and itching in his tongue.)

During the past two weeks, headache which has been constantly present, but especially marked at night, has been a particularly prominent complaint. It is localized chiefly in the left frontotemporal area. His head "feels drawn together very tightly." The intensity of the headache has been variable, but it has progressed recently to such an extent that it is almost unbearable. He has never been nauseated and never vomited; except for diplopia his vision has been unaffected. He does not think that he has lost weight. There have been no night sweats. His bowels are constipated. There is no nocturia. His sphincters are unaffected. He has never been dizzy, except when he attempts to use his eyes without his exclusion glass. His appetite is normal, although he does not taste as well as he formerly did.

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* Presented at the Clinical Conference, November 26, 1917

PHYSICAL EXAMINATION

Voluntary Motor System.—*Inspection* revealed a man well nourished and normally developed, but more or less haggard in expression and evidently suffering severe pain.

Abnormal Attitudes.—There were no abnormal attitudes.

Gait.—There was no alteration of the gait.

Coördination.—Coördination of the equilibratory and non-equilibratory types was normal throughout.

Skilled Acts.—Skilled acts were normally performed.

Reflexes (1 = slight, 2 = active):

Deep:

	Right		*Left*		*Right*		*Left*
Tricipital	1	=	1	Achilles	1	=	1
Bicipital	1	=	1	Abdominal	1	=	1
Supinator	1	=	1	Epigastric	1	=	1
Patellar	2	=	2	Cremasteric	1	=	1

	Right		*Left*
Plantar—Normal	1	=	1

I have indicated the deep reflexes as equal although at times I was suspicious that the right side was more active than the left. Repeated examinations, however, have not revealed sufficient difference to indicate any definite inequality.

Abnormal Involuntary Movements.—There were no involuntary movements.

Muscle Strength.—The myosthenic status was normal.

Muscle Status.—The volume, contour, consistency, and tone of all muscles were normal. Electrical reactions were not tested.

Abnormal Associated Movements. — There were no abnormal associated movements detected.

Nerve Status.—There was no abnormality found. (*See* sensory disturbance below.)

General Sensory.—The general sensory examination, including cotton wool, pin prick, and thermal tests, was negative except for the findings recorded below under Cranial Nerves. (Note the subjective sensory complaints of the patient.)

Cranial Nerves. *Olfactory.*—Smell was not tested.

Optic and Oculomotor Apparatus.—Vision was not examined. The fundi were entirely negative. (This finding was corroborated by Dr. Ward A. Holden on two occasions.) Visual fields were complete. The corneal reflexes were present. The pupils were round, central, slightly unequal, the right being 4 mm. in diameter, and the left 3 mm. They reacted briskly to light and accommodation. There was diplopia on looking to the right. There was a coarse nystagmus to the right, and a fine nystagmus to the left. Conjugate movements were normally performed except for a complete right external rectus paralysis.

Trigeminal and Mouth.—The motor fifth was normal. The sensory divisions showed marked changes. There was hypesthesia to cotton wool over the entire distribution on the right side. Hypalgesia was correspondingly found. The mucous membrane of the right cheek and gums was almost analgesic. There was marked diminution of sensibility to heat and cold compared with the left side.

Facial and Face.—There was no abnormality of contour of the face. I was unable to satisfy myself that there was any motor weakness on either side in any of the three divisions.

Taste.—Taste, however, was absent on the right side of the tongue, but as it was also very poorly determined on the left side, I have been unable to attach much importance to this finding.

Acoustic and Ear.—Hearing was normal on the left. A watch tick could be heard on the right only upon contact. To tuning fork tests bone conduction was greater than air on both sides.

The cold water Bárány test in the right ear produced a slow nystagmus to the right; in the left ear a slow nystagmus to the left.

Glossopharyngeal, Vagal, Pharynx, and Larynx.—The palatal and pharyngeal reflexes were present and normally performed. There was nothing abnormal observed in the pharynx. Voice was somewhat nasal in character. The vocal cords were not examined.

Spinal Accessory.—There was no abnormality of the trapezii or sternocleido-mastoids.

Hypoglossal and Tongue.—The tongue was protruded in the midline and presented no tremors or atrophy.

Mental Status.—Mental status was normal.

Cranial Morphology.—Cranial morphology to inspection and palpitation is normal. (*See* note on *x*-ray findings below.)

Systemic. *Tegumentary.*—There were no abnormalities detected in the skin or appendages. On the right there is a postoperative scar indicating the removal of posterior cervical glands. It is 1½ inches long and extends from the level of the angle of the jaw downward.

Glandular.—The glandular status is normal.

Cardiovascular.—The cardiovascular system is negative.

Respiratory.—The respiratory status is normal.

Gastrointestinal.—The gastrointestinal function is entirely negative. The bowels are somewhat constipated. There has never been any nausea or vomiting during this illness. There has never been any incontinence.

Genito-urinary.—Vesical and sexual control are entirely normal.

Skeletal.—There are no abnormalities.

Laboratory Tests. *Wassermann.*—The Wassermann reaction of the blood serum is negative.

Lumbar Puncture.—The cerebrospinal fluid was normal; being negative to the Wassermann reaction; with five lymphocytes, and one plus globulin.

Blood Count.—A differential blood determination was not made.

Urine.—Urinalysis was very thoroughly completed with negative results.

X-ray.—Up to the present no *x*-ray has been taken.

(*See note* below.)

SUMMARY

Summarizing the above pathological findings, we have first, headache localized in the left frontotemporal area, pain of a neuralgic character in the right temporal region, a paralysis of the right external rectus, diminished sensibility for all forms of sensation in the right trifacial distribution, deafness in the right ear, and nystagmus.

In considering the above findings, one is tempted to think first of the possibility of an intracranial tumor located somewhere in the posterior fossa on the right side. If one bears in mind the fact that the symptoms appeared one week after a surgical operation which was supposedly done for the removal of a tuberculous gland, it is easy to imagine the presence of a tuberculoma on the same side. However, inasmuch as we have been unable to detect any pyramidal irritation and no retinal disturbance, I do not feel that such a diagnosis is justified.

We cannot be dealing with a tuberculous meningitis, for the duration and history of the progress of the disease together with the spinal fluid findings have ruled it out.

I personally do not believe that syphilis can be considered a factor in this case.

Multiple sclerosis has often produced much more complex pictures than we have here, but I do not believe that it can be seriously considered in the absence of fundus, superficial, and deep reflex disturbances.

As I stated at the beginning, I am of the opinion that one cannot be definite as to the diagnosis or localization of the lesion in this case without further investigation from the standpoint of sinuses, *x*-ray data, and careful observation in the hospital. However, I felt that the case would present an opportunity for lively discussion as to the probable diagnosis, and I will be glad to report at a later conference the results of the patient's hospital experience.

DISCUSSION

Dr. O. S. Strong.—My impression is that this is a root lesion of some kind; secondly, that it is a meningeal involvement. The diagnosis of multiple sclerosis here does not appeal to me, particularly on account of the onset. It cannot be syringomyelia on account of the sensory changes. It is hard to see how the lesion hits the roots that way; it must include the ganglia roots along the cranial cavity. You would not get the symptom of dysesthesia so marked with the spinal fifth. It must be a root lesion involving these nerves and extending to the nerves mentioned. It might be due to tuberculoma or a local meningeal process. I would favor the diagnosis of a root lesion.

Dr. Norman Sharpe.—From the symptoms here presented, I would be inclined to put the lesion in the middle fossa about the ganglion of the fifth nerve, and probably extradural. As the first division of the fifth nerve is the one chiefly involved, it would seem that the lesion is a little anterior to the ganglion and not behind it. If it is contended that the seventh nerve is involved by a basal lesion, it would be placed further back in the posterior fossa, but in that case there would not be this character of pain. Sarcoma frequently lies in the median fossa and often around the fifth nerve, and in these cases the sixth nerve is very frequently the next to be involved. The sinuses should first be carefully examined.

Dr. Randal Hoyt.—It seems to me that in a case such as this, other than purely neurological factors should be taken into consideration. If the patient has had a middle-ear disease, or a frontal, antral, or sphenoidal sinus infection, this might account for the picture under consideration. The sixth nerve has the longest course of any within the cranial cavity and is usually the first to be affected in a basal infection. The fifth nerve, exposed as it is, is also extremely vulnerable to irritative agents, and is the first to make one suspicious on account of its definite and characteristic reaction. The condition of the excised gland may possibly have been secondary to an infective process and not tuberculous at all. Perhaps, as has been suggested, the lesion is a neoplasm, or a tuberculoma, but this is an important field and we should take into consideration the clinical picture which the aurist often sees and which means to him the extension of an infection from a sinus into the cranial cavity.

Dr. Michael Osnato.—I should like to recall to Dr. McKendree that I made an observation in regard to this case, when I first saw it, about the narrow palpebral fissure on the left side; there is a distinct difference in the two fissures. I think this is a tumor of the brain. If the patient had choked discs everyone here would agree with me, but it is quite possible to have a neoplasm of the brain without this sign, particularly if it is in the pons. Bing says of tuberculoma that forty per cent. never showed any eye changes in all their course. Here we have involvement of the right fifth, sixth, and seventh nerves, nystagmus and some indication in the caloric test that there is blocking of the vestibulocerebellar tract on the right side; there is spontaneous past pointing to the right and, with the cold water test, there was a more nearly normal past pointing than there should have been. I believe this man shows pyramidal tract signs on the right side. The difference in the reflexes points to left-sided pyramidal involvement. The left sensory changes in both seventh nerves and the possible involvement of the left seventh may be explained perhaps by a diffuse tuberculoma in the substance of the pons involving the fifth, sixth, and both sevenths, with lateral fillet involvement explaining the loss of hearing on both sides but which is more pronounced on the right. The absence of fundus findings does not change my belief that this is a lesion of the pons. As far as a meningitic process is concerned, this condition began nine weeks ago; tuberculous meningitis usually finishes up in two or three weeks and is invariably fatal. Syringobulbia cannot be considered nor can multiple sclerosis. I believe this man has a tuberculoma of the pons.

Dr. I. S. Wechsler.—I believe with Dr. Osnato that in this case we are dealing with a pontile lesion, probably a sarcoma.

Dr. C. A. McKendree.—In making an attempt to localize this lesion, the first suggestion was that of pontile involvement. Certainly, we have involvement of the fifth and sixth cranial nerves, but in order to establish the presence of seventh nerve paralysis we have to beg the question. I cannot attach importance to the sensory seventh findings as they were not confined to the one side. Nor have I been able to satisfy myself that the facial weakness which Dr. Osnato describes is a true observation. Therefore, in the absence of involvement of the seventh, and without evidence of involvement of the pyramidal fibers, I do not believe that we can consider a pontile lesion in this case.

As I have said before, I believe that our data are insufficient to establish a definite diagnosis. We must have careful investigation by the aurists and x-ray men before attempting to determine the nature of the lesion or to localize it.

SUPPLEMENTARY EXAMINATIONS

The following notes are transcribed from the records of the Presbyterian Hospital, and I am indebted to the observers there for permission to include their findings in this report.

LARYNGOSCOPIC EXAMINATION, *November 30, 1917:*

Septum thickened and irregular with a crest near the floor on the left and a deviation high up on the right. Considerable brown mucopurulent secretion on the floor of the left nares. Patient's frontals illuminate equally over a small area. The antra illuminate rather poorly; the left darker than the right. Both inferior turbinates are pale and edematous, with a bluish grey color (slate color). The oropharynx negative.

In the nasopharynx there is a mass situated at the vault about the size of half an English walnut, spongy in appearance with many minute blood vessels appearing on the surface. It looks not unlike adenoid tissue, but bleeds very easily. Gentlest application of a cotton applicator through the nose causes a hemorrhage from this mass. This mass nearly fills the mid-line, and to the left of the line. It covers one pharyngeal end of the Eustachian tube on the right. Larynx negative.

His left ear is negative except for moderate thickening of the drum and a slight amount of retraction. The right drum appears thickened. There is a loss of luster and an obliteration of the land marks. On the hearing test he hears ordinary conversation well at six feet with right ear, hears watch with right ear only on contact. Hearing normal on left. No impairment of nerve, but considerable impairment of sound-conducting apparatus. This may be due to the mass obstructing the tube in the nasopharynx. Would advise removing part of the mass for microscopical examination.

(*Signed*) E. L. PRATT

Above is correct. Mass apparently at base of sphenoid on right, but not coming through anterior wall.

(*Signed*) CORNELIUS G. COAKLEY

TUMOR OF NASOPHARYNX, *December 2, 1917:*

Gross Specimen.—Specimen is a mass of whitish tissue about 1 cm. in diameter which is soft friable and somewhat lobulated in architecture.

Microscopic Examination.—Surface of the tumor impinges directly upon an exceedingly thin layer of stratified squamous epithelium which in places is entirely wanting. It is made up of broad alveolar groups of closely packed, large, pale-staining epithelial cells, cuboidal in shape with a large pale-staining vesicular nucleus with, in many cases, from one to four dark-red staining rounded bodies which are, possibly, nucleoli. Mitotic figures appear on an average of one to every two or three high power fields. Between the alveolar masses are slender strands of connective tissue bearing blood vessels, and the whole mass is everywhere infiltrated with round and plasma cells with occasional leucocytes. The appearance of the individual cells and their arrangement strongly suggests a tumor arising from the anterior lobe of the hypophysis.

Diagnosis.—Carcinoma of Hypophysis.

(*Signed*) DR. PURDY STOUT

SECOND TUMOR MASS, *December 10, 1917:*

Tumor does not resemble any tumor arising from nasopharynx or neighboring sinuses. On the other hand, it is almost microscopically identical with a carcinoma that was known to arise in the hypophysis. It is on this basis alone that the diagnosis of a hypophysis tumor is suggested.

(*Signed*) DR. PURDY STOUT

X-ray of the skull at the Presbyterian Hospital did not reveal any definite involvement of the sella turcica although there were shadows indicating infiltration in that region.

Dr. Coakley recently operated upon the patient, exploring the ethmoidal and sphenoidal cells by the orbital route. The floor of the sella seemed normal and no communication between the sphenoidal cells and the surface of the sella could be made out. Tissue which was removed during the exploration proved to be definitely carcinomatous and of the same type as that removed from the vault of the nasopharynx which proves its origin.

During the patient's stay in the hospital, I am informed that the neurological features remained unchanged. He has recently been discharged in practically the same condition as he was when admitted. The prognosis in his case is, of course, hopeless.

DYSTONIA MUSCULORUM*

By MICHAEL OSNATO, M.D.
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This patient is presented for two reasons: first, because of the extreme difficulties which he has offered for diagnosis; and second, as a timely warning that a diagnosis of functional disturbance—particularly hysteria—should be made only as a result of a careful study of the case and by a process of elimination.

I have carefully examined this patient numerous times during my year of service in the wards of the Kings County Hospital, and have taken particular pains to observe him on the occasion of each of my visits. When I first saw him I was told that he was a case of habit spasm and was essentially a neurosis. Although repeated examinations left me puzzled, I was sure of one thing, namely, that this was not like any case of hysteria or neurasthenia I had ever seen. I considered it at first a chronic chorea, but after careful study—particularly after reading J. Ramsay Hunt's report of seven cases—I concluded that I dealt with a case of dystonia as first described by Ziehen, seven years ago.

A brief review of the work of Ziehen and Oppenheim in Europe, and Frankel and Hunt in this country seems apropos. Ziehen regarded the affection as a neurosis and proposed the name *tonic torsion neurosis*. The chief characteristics of his cases were progressive spasms and hypertonicity of the musculature with twitching movements of the extremities and trunk, increased by active movement and diminished during rest. The gait and station were chiefly affected and there was a lordosis or kyphoscoliosis of the spine. All of the patients were Russian Jews. He compared the movements to those of chorea, athetosis, and hysteria, but allied his cases to the tics. Oppenheim, a year later, considered his cases in the light of an organic disease of the nervous system allied to athetosis. All of his cases were also Russian or Polish Jews and began, like Ziehen's cases, between the ages of eight and fourteen. According to Oppenheim, the spasm began in one of the extremities, either the leg or the arm, but gradually all the muscles of the thigh, pelvis, and spinal column were chiefly affected. He mentioned the lordosis and described the gait as a dromedary or monkey gait. He also particularly noted the tendency to clonic and rhythmic movements, mobile spasms, and in some cases tremor of the paralysis agitans type. He too compared his cases with chronic chorea. Oppenheim laid particular stress on the tonic muscular spasms in groups of muscles with twitching and torsion movements in the spine and pelvis as the dominant features and stated that they were associated with hypotonus in opposing groups of muscles. On the strength of this alternating hypotonus and hypertonus Oppenheim postulated the opinion that it was an organic disease due to a disturbance in the mechanism which controls muscle tonus. His particular descriptive name for the disease was *dysbasia lordotica progressiva*. In the affected group of muscles the tendon reflexes were diminished by the hypotonia.

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* Presented at the Clinical Conference, November 12, 1917

Frankel, in differentiating it from athetosis, pointed out the localization of the spasm in the trunk, neck, and pelvis as against athetosis, which occurs more particularly in the extremities. Before the New York Neurological Society in 1912, Frankel showed four cases, two of which form part of the basis of Hunt's study of seven cases. He grouped his cases with the tics, and suggested the name *torti pelvis*. The only case on record which did not occur in one of Jewish parentage is one reported by Spiller of this country. This case was particularly noteworthy because it was familial, two sisters being affected, and all three were mentally defective.

All of the authors described remissions of varying duration but agree that it is essentially a progressive disease. They all seem to agree also that the spasm never attacks any of the muscles supplied by the cranial nerves, if one does not consider the eleventh as a cranial nerve. None of them have described disturbances of sensation or of the pyramidal tracts.

J. Ramsay Hunt, in a discussion of the subject,[1] also lays particular stress on the point that the spasm does not occur in the distribution of any of the cranial nerves, and he is inclined to doubt the diagnosis in any case presenting spasmodic movements in muscles supplied by those nerves. He confirms the findings of Oppenheim, and agrees that it is an organic affection of the central nervous system. He draws attention to the absence of hysterical stigmata and to the mental characteristics of the patients who suffer from convulsive tic as evidence against allying this clinical entity with either of those conditions. He suggests that the anatomical basis for the disease may be found in the destruction of the large multipolar cells found in the corpus striatum, in all three of its subdivisions, namely, the globus pallidus, the putamen, and the caudate nucleus, and their connections with the red nucleus, the subthalamic body, and the substantia nigra. To the small multipolar cells found in the corpus striatum, Hunt attributes the functions which are disturbed as a result of Huntington's chorea, the Vogt syndrome and Wilson's disease. He regards the large cells as predominantly motor and the smaller cells as coördinating and inhibitory over automatic and associated movements. He therefore places this disease in part in the corpus striatum, because of its close relationship to athetosis and chorea. He points to the fact that the muscles of facial expression and articulation are not involved in this disease as an argument against the absolute localization of the disease in the corpus striatum. He thinks that the connections of this body with some of the little known bodies such as the subthalamic and the substantia nigra are at fault, and he ascribes to this mechanism the regulation of tone, disturbances of which he thinks are the prime factor in the production of this disease. Hunt seems to have found a distinctive symptom in these cases, particularly in advanced cases which are bedridden and contractured, the curious reversal of the normal tonus manifestations, which he regards as pathognomonic. He calls this the paradoxical phenomenon of dystonia, which is seen in the ankle, less often in the wrist, and which consists in a disturbance 'of the voluntary control of tonus in the affected muscle groups'. The test is brought out by asking the patient to perform plantar flexion of the foot when it is in dorsal spasm; this results in a further increase in the extensor spasm instead of the normally executed plantar flexion. This, even though the command is understood and the effort made, is explained on the ground that the antagonist, which is in a state of hypertonia, resists the impulse sent out to it. With this preliminary review of the work done in this type of cases, I present this patient.

Patient: F. J. M.; age 38; male; single; clerk. Admitted to the Kings County Hospital in the fall of 1915—almost two years ago.

HISTORY. *Present Illness, Beginning with Complaint.*—He complains of spasms and convulsive movements in the muscles of his back, trunk, and neck, and of a deformity of the spine which makes it impossible for him to assume an erect attitude. He states that when he is quiet in bed the spasm is greatly relieved and it disappears at times during sleep, but the minute he gets up to walk the spasm increases enormously and he twitches and squirms. He has not been able to stand up straight for many years. The patient denies venereal infection.

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[1] *Am. M. J.*, Nov. 11, 1916

Previous History.—At the age of ten years the patient had an operation for circumcision. About the same time the patient's father became ill and died suddenly. He was left alone for a long time with his dead parent and became greatly frightened. Soon after that he was treated by his family physician for what the patient describes as nocturnal emissions, and suddenly during the treatment developed an attack of twitching in the neck and trunk which was called St. Vitus's dance. This choreiform attack was so persistent and yielded so poorly to all forms of treatment that he was finally advised to go to the Hospital for the Ruptured and Crippled where he remained for more than a year. During that time he received the ordinary treatment for chorea which consisted of Fowler's solution, etc., with very little benefit. At the end of a year's stay in the hospital, however, he improved considerably; the twitching in the neck and trunk was only slight, so that he was able to return home. He went to work, and the occasional slight twitching did not interfere with his occupation until the age of eighteen when he had a sudden exacerbation which was extremely violent and was ascribed to masturbation and the abuse of tobacco. The spasm in the neck on the right side was so severe at the end of three months that he had to go to St. Mary's Hospital in Brooklyn where he was kept in bed for seven months. He remained at the hospital thirteen months altogether. The right side of his neck became so swollen and tense if he got up that the physicians of the institution insisted on his staying in bed for the entire seven months which he mentions. Gradually the spasm grew less, however, and he was discharged again, improved, although there was still some twitching in the neck and trunk when he left St. Mary's Hospital. The treatment consisted of rest in bed, Fowler's solution, iron, bromides, etc.

Following this, he began to study after working all day, and kept at it pretty well. At twenty-four years of age he had another attack of neck and trunk spasm which was severe for only a few months. He had occasional twitchings and spasms until he was thirty-six years of age, but at this time the torsion spasm became so pronounced that he had to quit work and, after attending the Brooklyn Hospital, the Vanderbilt Clinic, and other clinics, was finally admitted to the Kings County Hospital about two years ago. The patient states that his malady had been diagnosed variously as habit spasm, chronic chorea, tic, hysteria, etc. No one has ever been able to relieve him.

Family History.—The patient's father died suddenly at the age of thirty-one of heart disease. His mother is alive and very active at the age of sixty; she has never been ill. There were three children in all; one was a stillbirth and one died of diarrhea in infancy. It is stated by the mother that no member of the family ever had an affliction similar to the patient's, and that there has been no insanity, epilepsy, or nervous disease in the family so far as she knows.

GENERAL EXAMINATION

Inspection shows a thin, poorly nourished individual who cannot stand quiet, his body inclining to the left. There are fairly rhythmical twitching torsion movements of the trunk, pelvis, and the left upper extremity, and particularly of the neck. The neck is spasmodically drawn to the left, the chin pointing sharply towards the left shoulder and the head being drawn backward. There is no spasm of the muscles of the face. The back shows a considerable prominence on the right side of the chest, due to a lordoscoliosis to the right with a corresponding hollow in the left side of the trunk and chest. The curve takes in the lumbar and all of the dorsal region; the whole body, including the trunk, shoulder, head, and neck inclines considerably to the left. The torsion twitching movements in the neck and trunk are much more marked when the patient walks than when he lies down. There are also twitching choreiform athetoid-like movements, particularly marked in the left upper extremity.

Mensuration shows no difference in the corresponding parts of the upper and lower extremities but the left chest is two inches less in circumference than the right due to the deformity.

Palpation and Massage show very distinctly alternating spasm and relaxation of the left sternocleidomastoid and the muscles of the left upper extremity and trunk, and also disclose the hollow in the left side of the chest posteriorly and a very distinct scoliosis and lordosis in the dorsolumbar spine.

SPECIAL EXAMINATION

VOLUNTARY MOTOR SYSTEM. *Gait.*—The gait is somewhat stiff and hurried but not spastic. In walking the torsion spasm in the neck, trunk, and in both arms becomes particularly marked, the patient gyrating and twisting with the whole body including the head, leaning decidedly to the left. It appears at times when the patient walks as if he were about to fall over. He makes a very distressing appearance.

Coördination.—Coördination, tested with the eyes open and closed, standing and lying down, with the finger-to-finger, finger-to-nose, past-pointing, knee-to-heel, knee-to-heel-to-toe and toe-to-finger tests, is normal and equal on both sides. The tests are greatly interfered with, however, by the sudden turning and twisting movements of the trunk, neck, and left upper extremity with which are associated also irregular involuntary movements of the lower extremities which, however, are not really part of the spasm. At the end of some of the movements in the finger-to-finger and finger-to-nose tests, the index finger of the right upper extremity wavers irregularly in a tremorlike manner somewhat suggestive of the intention type. It is extremely difficult to say whether this is a real tremor or whether it is an irregular movement associated with and caused by the torsion spasm. There is marked adiadochocinesis in the left upper extremity.

Skilled Acts.—The performance of skilled acts is interfered with only by the spasm, and I have often wondered how it was possible for the patient to undress and dress. He is able to write fairly well despite his spasm. His speech is normal excepting when his torsion spasm is very great, when it becomes jerky and snappy as a result. This, however, must be understood as an incident and not primarily as a speech defect.

Reflexes.—The reflexes in the upper extremities are usually hard to elicit—especially the pectorals, the triceps, and the biceps. The jaw jerks are very much diminished; the patellars are fairly active but equal; the superficials are all present and equal. The reflex table (0 = absent, 1 = slight, 2 = active) follows:

	Right		*Left*
Pectorals	1	=	1
Triceps	2	=	2
Biceps	R	=	L
Prepatellar	R	=	L
Patellar	2+	=	2+
Ankle	1+	=	1+
Dorsocuboidal	R	=	L
Upper abdominals	R	=	L
Lower abdominals	R	=	L
Cremasterics	R	=	L
Epigastric and suprapubic	Present		

There is no Babinski and none of the modifications, and no clonus.

Muscle Strength.—Muscle strength in this patient is extremely hard to test in the upper extremities because of the intense spasm; I should say that it was normal, however. In the legs the muscle strength is also normal. In the neck it was greatly interfered with by the spasm and could not properly be judged. The left sternocleidomastoid seemed to be hypertrophied as a result of many year's spasm, the right also appeared more prominent.

Muscle Status.—Muscle Tonus: There is a general marked hypotonus in all the muscles of the patient's body. The only point at which hypertonus is discernable is over the left sternocleidomastoid, and it alternates quite distinctly with periods of hypotonus. There is also an occasional spasmodic contracture of the right sternocleidomastoid with hypertonus which also alternates with hypotonus.

Myotatic Irritability: Shows no change to mechanical stimulation; there are no spontaneous twitchings in the muscles.

Myoedema: Is absent, the enlargement of the sternocleidomastoids being hypertrophic and not edematous.

Abnormal Associated Movements.—Appear in connection with the torsion spasm of the trunk and neck in both upper extremities, but more particularly the left. The movements here were more nearly athetoid than choreiform.

NERVE STATUS. *Electrical Reactions.*—At various times there has been quite consistently present the reaction which Oppenheim described in some of his cases as the dystonia reaction. In the muscles which take part in the spasm, particularly those of the neck, trunk, and left arm, it has been possible by faradic stimulation to cause active strong responses with the first seven or eight breaks of the current which gradually grew weaker and finally ceased altogether. After a rest of a minute or so, stimulation again produced strong contractions which became increasingly weaker with each successive stimulation. This was particularly well brought out in the left sternocleidomastoid muscle. It seems to me that this reaction particularly stamps this disease as not functional. *There is no nerve hyperexcitability.*

GENERAL SENSORY EXAMINATION. *Touch, Light Touch, Pain, Temperature, Vibration, Pressure, Spatial Localization, etc.*—All normal.

CRANIAL NERVES.—All the cranial nerves are normal.

Optic and Oculomotor Apparatus.—The pupillary reactions, both to light and accommodation, consensually and in convergence, are physiological and equal. There is no nystagmus; no strabismus. There are no sensory changes in either the fifth or seventh. The fundi are normal.

Olfactory and Nose.—Smell is normal.

Acoustic and Ear.—Hearing is normal.

Hypoglossus and Tongue.—There is no deviation of the uvula or of the tongue.

MENTAL STATUS.—Normal. The patient is keen, alert, well educated, and frank. The cranial morphology is normal. Caloric Bárány tests are normal.

SYSTEMIC. *Cardiovascular.*—The cardiovascular status is normal except that the hands and feet are usually moist and cold and show evidences of a poor peripheral circulation, being prone to become rather bluish on exposure to cold.

Respiratory.—Normal.

Gastro-intestinal.—Normal.

Genito-urinary.—There is no disturbance in the rectal, vesical, and sexual control.

LABORATORY TESTS.—*Serology* is negative, both as to blood and spinal fluid.

Urine.—Normal.

X-ray.—*X*-ray examinations have disclosed nothing except the deformity of the spine.

SUMMARY AND INTERPRETATION

Apparently we deal with an extra-pyramidal disease without changes in sensation, gradually progressive, with remissions, not occurring in a Hebrew, and without familial or hereditary taint. The disease began at the age of ten and has been characterized by a peculiarly persistent torsion spasm of the neck and trunk with associated movements in the left upper extremity and by a gradually increasing deformity consisting of a lordo-scoliosis in the dorso-lumbar region. The spasm has been characterized by a constant alternating change between hypertonus and hypotonus in the affected muscles, not influenced by treatment of any kind.

THE DIAGNOSIS.—1. *Symptomatic:* Progressive torsion spasm.

2. *Anatomical:* Not made.

3. *Etiological:* In my opinion an organic nervous disease of the central nervous system, as a result of destruction of that portion which has to do with the maintenance of tone.

DR. S. P. GOODHART.—Whether this is a case of dystonia musculorum deformans, a clinical entity originally described by Oppenheim, or whether it is one of those which we place among the so-called tics, it is certainly one of unusual interest. The likelihood of its being a type of hysteria, in other words, purely functional, is most remote. I have had under observation for the last two years two cases which were conceded to be dystonia when finally studied at the Montefiore Hospital, both females in early adult life, who had been dealt with in other institutions as cases of purely functional character.

The fact that this man is not a Hebrew is not against the diagnosis of dystonia musculorum deformans; we can no longer regard these cases as occurring solely among Russian Jews, as since Oppenheim's original report cases have been found among other races.

In the cases which I studied at Montefiore, the essential feature, that is, the torsion of the trunk about the pelvis in locomotion, was distinctly manifest; in the case before us that dominant characteristic of the disease is only slightly to be observed. The hyper- and hypotonia which in my experience alternate even in the same muscles from time to time is by no means distinguishable here. A feature present here which I do not believe characterizes the typical syndrome is what appears to be the rhythmical movements of the head; the sternocleidomastoid and trapezius alone seem to be involved in the rhythmical contractions resulting in the head movements. In true dystonia these movements are less rhythmical and more variable.

In one of the cases at Montefiore, and of unusual interest, there were periods, varying in time but occasionally covering several weeks, in which there were very marked exacerbations of all the movements, the hypertonia of the back and the head being so tense as to cause extreme pain, sometimes for hours throwing the patient into the position of opisthotonos. Synchronous with this hypertonicity there was marked increase in tone of the hamstring muscles of one leg, developing for a period of time to such contracture that the leg was semiflexed—a position which could not be relieved even by deep anesthesia. In this patient there were likewise periods of tachycardia sometimes lasting for hours which, together with the extreme muscular tension and activity, threatened life by exhaustion.

While the case before us is not to my mind a typical dystonia, I am strongly inclined to believe the condition is due to an organic lesion, and probably its pathology will be found in that region where the basal ganglia alone seem to give us the interpretation of these abnormal movements.

Dr. I. S. Wechsler.—I have seen only one case of typical dystonia musculorum, but although it had taken a much shorter time than this one to develop, it was more marked than the case shown here, which seems to have gone on for more than twenty-five years. It seems to me, if this case is a true dystonia musculorum, it ought to have progressed to greater deformities. As I understand it the classical picture shows sternocleidomastoid involvement and a marked kyphoscoliosis, and in addition, one of the cardinal symptoms, as Dr. Goodhart pointed out, is the torsion of the spine and the turning on itself of the pelvis. None of these signs are at all marked or even present in this case. The dystonia too is more rhythmical here than one would expect and there are no athetoid movements. While the case is very interesting and suggestive of dystonia musculorum progressiva, one should bear in mind a possible functional disorder.

Dr. F. M. Hallock.—From the description of dystonia musculorum in the literature, this does not seem to me to be a typical case. The only one I have ever had the opportunity of studying over a length of time, however, was not exactly typical of the syndrome. This case appears to me to be organic in origin and not functional. The pelvic deformity is certainly not present, and I do not see that this patient exhibits the peculiar athetoid and choreiform movements that belong with the syndrome. In addition, I am not satisfied that the hypotonia, to which Dr. Osnato calls attention, has been demonstrated. Altogether the case strikes me as apart from the dystonia musculorum group and seems to belong in some other class of abnormal hypertonicity, possibly related to the tics.

Dr. O. S. Strong.—To the uninitiated this case might appear like a mélange of chorea, athetosis, some kind of spasm and multiple sclerosis with a dash of paralysis agitans.

CLINICAL OBSERVATION ON SENSORY CHANGES IN TRAUMATIC NEURITIDES FROM THE STANDPOINT OF PROTOPATHIC AND EPICRITIC SENSIBILITY*

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Several months ago Dr. Osnato † showed a case of extreme hyperalgesia and hyperesthesia involving the ilio-inguinal nerve, due to its inclusion in a suture introduced for the purpose of fixing a floating kidney. What interested the writer at the time was the fact that there was no increase nor any change in the thermesthetic sense. A question at once presented itself: If the conception of Dr. Head and his co-workers as to peripheral sensations is correct, why did not the above case show a hypersensitiveness to temperature as it did to touch and pain? Feeling that so much brilliant and painstaking work had already been done by other and more competent men, the writer was loath to undertake the study. The subject was approached, therefore, with some misgiving as to the importance of the findings likely to result from such clinical investigation. After several months' continued observations, however, the results obtained seem sufficiently important to deserve recording as a preliminary to a later, more extensive contribution.

It will be recalled that Dr. Head and his collaborators [1] have divided peripheral sensibility into deep, epicritic, and protopathic. Under the first they include pressure and its localization, pressure pain, movements of joints, muscles and tendons, their extent and direction, *i.e.*, position. Under the second are included tactile discrimination (compass points), light touch and its localization, and temperatures above 20° C. and below 40° C. Under the last they included temperatures below 20° C. and above 45° C. and all cutaneous pain. Head claims that only protopathic and epicritic sensation is carried by the superficial sensory nerves, all deep sensation being carried by afferent fibers running in the motor nerves. On the last point he quotes Sherrington [2] who demonstrated the presence of afferent fibers in motor nerves.

The two systems of cutaneous sensation, *i.e.*, epicritic and protopathic, are distinct, according to Head. Whenever a purely sensory nerve is completely severed there is total loss of all superficial sensation. But the area of loss of protopathic sensibility is much smaller than that involving the epicritic. The latter represents the anatomical distribution of the nerve and shows distinct delimitation, while the former is less than the anatomical distribution, showing overlapping by anastomosis of the end twigs. In every case of nerve injury, therefore, Head maintains that there always is an area of complete loss of epicritic sensation and only a part of this area shows protopathic loss. This leaves an 'intermediate zone' which can perceive pain and extremes of temperature, but shows no response to touch and other epicritic stimuli. The recovery of protopathic sensibility proceeds much faster and often is complete months before the epicritic has begun to show signs of improvement. This, it is claimed, is true in all cases where the nerve has been completely severed.

Careful and repeated clinical observation extending over a period of many months failed to confirm all of Head's findings. The methods of testing employed were those useful for clinical purposes as contrasted with those of the laboratory. For touch, cotton wool was used; for pain, a sharp pin or needle; for temperature, narrow test tubes filled with hot and cold water; for tactile discrimination, a dull-pointed compass; for deep pressure, the eraser end of a pencil; and for vibratory sensation, a tuning fork, 128 p. m.

Case I.—L. R. cut his right wrist with a piece of glass on November 1, 1916, dividing all the flexor tendons (except the flexor carpi ulnaris and the flexors of the thumb) and the median and ulnar nerves. The tendons and nerves were sutured twenty-four hours after the injury, and the wound healed by primary union. Recovery was slow but progressive, and Figure 1 shows the sensory status on July 3, 1917.

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† Presented at a clinical conference of the Neurological Staff of the Vanderbilt Clinic

* From the Department of Neurology, Columbia University

The shaded areas represent those parts which showed altered sensibility. In the area shaded with vertical lines there was hyperalgesia, hyperesthesia, tactile discrimination (circles of Weber) at 1 cm.; pressure pain more acute, tactile and pressure localization normal. Cold, both epicritic and protopathic delayed. In area shaded with horizontal lines: above dotted line hyperalgesia and hyperesthesia; below dotted line hypalgesia and hypesthesia. For entire area tactile discrimination completely lost; tactile and pressure localization fairly accurate; pressure pain diminished. Both epicritic and protopathic temperature diminished and delayed. Position sensation normal; vibratory sensation diminished. September 29, 1917, light touch perceived all over, pin prick perceived all over; there was slight hyperalgesia; tactile discrimination still lost in area shaded with horizontal lines; pressure pain normal, tactile and pressure localization normal, protopathic cold and heat diminished, epicritic heat and cold doubtful; position sensation normal; vibratory diminished.

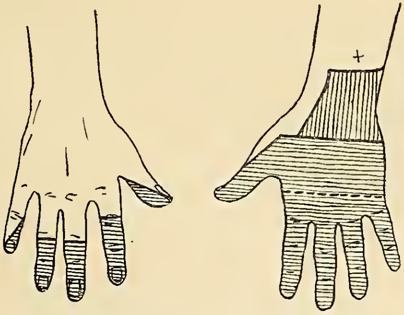

Fig. 1. Sensory Status of Case I, July 3, 1917
Cross indicates scar where injury occurred

Case II.—V. C., April, 1916, sustained a fracture of the left humerus. A plaster cast left in place for a month induced ulceration on the outer surface of the left forearm, resulting in numbness of left hand. There was inability to pronate the left wrist; loss of power in the thumb, index, and middle fingers; no electrical response in the flexors of those three fingers; all extensors normal.

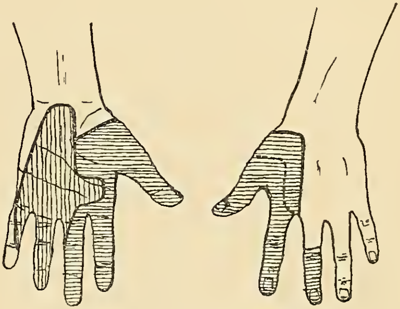

Fig. 2. Showing Sensory Status of Case II

July 7, 1917: Dorsal surface, loss of sensation to cotton wool and pin prick; loss of discrimination; loss of vibratory sensation; pressure localization normal; temperature, both epicritic and protopathic, lost; position sensation normal. Palmar surface, in area shaded with horizontal lines there was complete analgesia, complete anesthesia, complete loss of temperature sensation; loss of vibratory sensation; no tactile discrimination or localization. In area shaded with vertical lines, slight hyperalgesia, slight hypesthesia to cotton wool; diminished temperature sensation both epicritic and protopathic, cold being less perceived; tactile discrimination normal (1 cm.); pressure pain normal; pressure localization normal. November 3, 1917: Same findings except that on dorsal surface the normal sensory skin area extended to dotted line.

Case III.—M. C., April 12, 1917, had a general infection of the right hand which necessitated numerous incisions.

At the first examination, September 12, 1917 (Figure 3), there was loss of sensation to cotton wool and to pin prick in all shaded areas; absence of tactile localization and discrimination; pressure localization present; perception of all degrees of heat lost; vibratory sensation present. November 1, 1917: Same findings as above except that pain was felt in areas shaded with horizontal lines. Touch to cotton wool over hairs in this area was perceived.

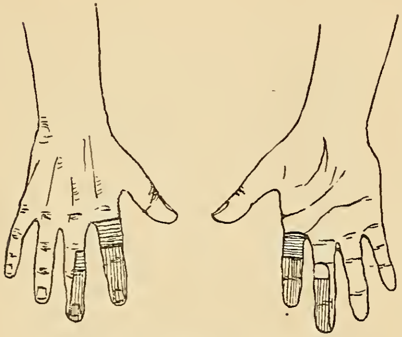

Fig. 3. Showing Sensory Status of Case III

Case IV.—J. K., June 10, 1917, received bullet wound through the right arm, severing the musculospiral nerve, resulting in wrist drop. Nerve was sutured.

The first examination, September 15, 1917, and repeated examinations thereafter, showed in area outlined in Figure 4: loss of sensation to cotton wool and pin prick; tactile discrimination, varying from 3 cm. to 5 cm. as indicated in the figure; tactile localization absent; pressure localization and pressure pain present; perception of all degrees of temperature absent.

An analysis of the findings in the foregoing cases does not confirm the conclusions of Head and his co-workers. In no case was there a distinct 'intermediate zone' in which there was loss of the so-called epicritic sensation and preservation of the protopathic. There was no direct relation between tactile localization and tactile discrimination. Case I showed the presence of touch and loss of discrimination. Case II showed distinct hypesthesia to cotton wool, yet normal tactile discrimination. Wherever sensation for mean temperatures was lost, perception of extremes also was lost.

In all the cases attempt was made to determine exactly where normal sensation ceased and where abnormal sensation began. Within the area of altered sensibility repeated attempts were made to determine whether there were any areas which showed preservation of pain and loss of touch to cotton wool, but it was invariably found that the two went together. Usually toward the periphery of the area perception of all sensations was only partially lost.

Deep sensation, *i.e.*, position, vibratory and pressure sensation, was found to be preserved in all cases where only cutaneous nerves were involved. This tends to confirm Head's contention that all deep sensibility is carried by way of motor fibers. In view of the established fact that the skin contains punctate sensibility [3] and specific receptors for every sensation,[4] no attempt was made to chart the different points of sensibility.

Trotter and Davies [5] in their experimental studies in the innervation of the skin also were unable to confirm Head's classification of peripheral sensations. Dana [6] in an examination of peripheral sensation of the cranial nerves (particularly the fifth) came to the conclusion that they showed nothing like

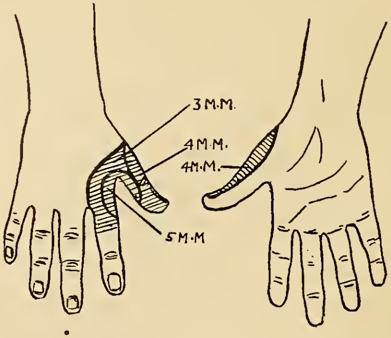

Fig. 4. Showing Sensory Status of Case IV

Head's differentiation into protopathic and epicritic sensibility.

Head maintained that only in the case of sutured nerves did the return of sensibility show division into epicritic and protopathic, but if there really are two different systems, how is it that they are disturbed only in the case of complete severance of the nerve? W. A. Osborne and B. Kilington [7] in their study on nerve fibers in regenerated nerves found distortion of sensory and motor patterns in sutured nerves but none in case the nerve was only crushed. Is it not possible that the difference in the sensibility is due to this distortion? Further, the rigid classification of peripheral sensation into protopathic and epicritic seems to be based on the assumption that there are different sensory receptors and different fibers for the perception of different degrees and modes of the same sensation. This assumption so far has not been sustained either by histological or physiological investigations. As a matter of fact, Head himself [8] has demonstrated that in the cord there is no differentiation into protopathic and epicritic, only he explains this on the theory that the peripheral sensations are more complex than those in the cord, and that the former are regrouped before they proceed into the secondary sensory levels.

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EPILEPSY WITH POSTNEURITIC OPTIC ATROPHY*

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I thought it might be interesting to present today a case that has attended the clinic for some time and has received material benefit from a disease which has existed for some years. It is of interest from three aspects: the question of etiology, the relationship of the condition to the functional background, and also the results obtained by therapy.

Patient: Mrs. F.; age 41; married.

History. *Family History.*—The family history is negative except for 'nervousness' in some of its members.

Previous History.—Her own previous history is also negative except that she has always been more or less excitable, sometimes irritable under just provocation, somewhat temperamental, and occasionally depressed. She has had no miscarriages, except one that was artificially produced.

Present Illness.—The illness began nine years ago, when she had what was called 'nervous prostration', manifesting itself at the beginning in violent headache and delirium to a point necessitating restraint, during which she became totally blind and deaf. Her sister assures me that it was necessary to shout in her ear to make her hear what was said to her. She became unconscious after a few days and remained so for two months. This attack followed great anxiety of a family nature. She gradually recovered from this illness and became, to all appearances, well.

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* Presented at the Clinical Conference, August 16, 1917

Suddenly, seven years ago (two years after the period of delirium and unconsciousness) she had an attack of convulsions in the night, which was, judging from all that I can learn, an attack of true epilepsy, with frothing at the mouth, relaxed sphincters, headache, somnolence, etc. Up to the time of her admission here she had had a similar attack about every six weeks, always nocturnal in occurrence.

She had no aura. She has headache always after her convulsions; on one occasion she was unconscious for two days after an attack, according to her statement.

She came to the Vanderbilt Clinic in October, 1915. Examination revealed the following:

VOLUNTARY MOTOR SYSTEM. *Gait.*—Normal.

Coördination.—The equilibratory functions were normal. There was no ataxia.

Reflexes (*Deep*).—Normal and equal.

(*Superficial*).—Normal.

Muscle Status.—Motoricity was found normal.

NERVE STATUS.—There was no tremor.

CRANIAL NERVES AND HEAD.—At this examination the cranial nerves were found to be normal.

SYSTEMIC. *Cardiovascular.* — Examination revealed nothing systemic except from the cardiovascular standpoint, the systolic blood pressure being 160. It has since fallen to 140 at times, but she still has a little cardiovascular hypertension.

LABORATORY TESTS. *Wassermann.*—The blood was found negative. The cerebrospinal fluid had 14 cells and was negative to the Wassermann test in 2 cubic centimeters.

Urine.—Two complete urinalyses proved negative except for faint traces of albumin.

In view of the above findings and because of the large hysterical factor which I found to be present—provoked for the most part by disturbances at home, financial disappointments, etc.—I was inclined to believe the epileptic attacks psychogenic in origin. But at a later visit careful examination of the fundi revealed an old postneuritic atrophy at both sides without elevation of the discs. The left disc was quite pale, the right slightly so. There was a slight concentric contraction of the right visual field. On the left, there was loss of the nasal half, which I believe is the usual field of postneuritic atrophy.

The presence of postneuritic atrophy of the optic discs clearly stamped the origin of the epileptic attacks as organic in nature. Were we dealing with the sequelæ of an encephalitis or of a basal meningitis, and what was the etiology? The history of deafness and blindness, followed by such a long period of unconsciousness with gradual clearing up of all except the ocular symptoms, seemed to be of unusual interest.

Treatment has consisted of low proteid diet, free elimination, regulation of the daily life, and luminal—one grain three times a day, for the past two years. The result is that she has had only six attacks in that time, pursues a daily occupation which was impossible previously, and her disposition has changed remarkably for the better.

I present this case for discussion of the etiological factors involved, and the probable pathology that accounts for the organic epileptic manifestation.

DISCUSSION

DR. I. S. WECHSLER.—The case seems to be interesting in that it brings up for diagnosis hysterical versus epileptic convulsions. But the sudden blindness and deafness following excitement or worry would make one think of psychogenetic etiology. As to the possibility of meningitis, the history does not show it, but there may have been some edema lasting that long with or without an actual inflammatory condition. The postneuritic atrophy may be explained by the pressure caused by the edema. The improvement is very gratifying; I know that luminal is one of the few drugs capable of causing it. I think the possibility of edema of the meninges should be considered.

Dr. Osnato.—I should like to ask Dr. McKendree to give us more definite information about the status of the patient during the period following the initial convulsion and during the two months of unconsciousness.

Dr. McKendree.—The initial convulsion came on two years after the period of unconsciousness and delirium.

Dr. Osnato.—During the two months of unconsciousness what observations were made?

Patient.—Perhaps I can best answer that myself from what I was told by my nurse. I was totally unconscious to all surroundings but suffered from the most intense headache constantly. There was no vomiting. Nutrition was taken through a glass vessel.

Dr. Osnato.—It seems hard to escape a certain conclusion in this case. We have one definite thing to work from, and that is the postneuritic optic atrophy; and I am inclined to believe, regardless of the negative serology, that we have to deal here with a specific luetic meningitis of the basilar type. The prolonged headache occurring over a period of two months, without vomiting or temperature, also the history of deafness and blindness—all of which have cleared up fairly well—point to a meningitis of the type usually seen in syphilis. It seems to me that we have to deal here with epileptiform seizures which are a residual symptom following the meningitis, and that the optic atrophy is postneuritic. It may be that certain psychogenic things in this patient may or may not have their influence in making the epileptic attacks frequent or infrequent; but I certainly believe that this is an organic case, the diagnosis being epilepsy due to a basilar meningitis with postneuritic optic atrophy due to syphilis.

Dr. Tilney.—This case interests me extremely. I do not like to take the time of the Conference in too often expressing my own opinion, but I feel justified in this case because it is almost exactly the same as one I have been following for the last three years and whose improvement, if I may say so, has been even more pronounced.

In the question as to the pathology, I think we have three things to consider in differential diagnosis:

1. Is this a psychogenic, or functional convulsion type of disorder?

2. Does it belong to the idiopathic group of epileptics?

3. Is it a symptomatic epilepsy?

Such organic evidence as persists to the present time is an argument against its being a functional disorder. Surely, the coincidence of so marked a change in the eye grounds with this sort of seizure would be most unusual; so we may rule that consideration out.

As to idiopathic epilepsy, I think this entirely improbable. This patient has none of the features of that type of case. She gives a history of onset too definitely symptomatic to allow it to be grouped in that class of convulsive seizures.

It seems to me to be a symptomatic epilepsy. With that before us, we must decide what etiological factor could produce such a pathologic change or alteration in brain structure. Dr. Osnato's suggestion is the first to be considered; a syphilitic basilar meningitis. It is not the only possibility, however. Another consideration is a condition which is too little regarded in these situations, namely an ependymitis, *i.e.*, an inflammation of the lining of the ventricles or of the aqueduct with choked disc and secondary optic atrophy; also inflammation of the brain tissue itself—encephalitis. Many cases of encephalitis have escaped attention as such, but belong to a definite group of polioencephalitis, and I believe this woman may have suffered from an infection of the organism of Flexner and Noguchi—the globoid bodies.

The patient I saw was similar in many respects. Eight weeks after the birth of a child she became septic. Gradually she began to show mental symptoms and suddenly went into prolonged coma in which she remained for some weeks, then emerged both deaf and blind with definite choked discs in both eyes. Six weeks after the appearance of these latter symptoms she developed generalized convulsions unlike the ordinary idiopathic epileptic convulsions, the seizures lasting over an hour. Following this she had seven convulsive attacks in rapid succession. She came to me after many convulsions—having them at the rate of one a month—and I sent her to Roosevelt Hospital for more careful observation. We went over all the possibilities of her case. The spinal fluid and blood were negative. The urine and feces showed nothing. There were no abnormalities of the intestinal canal. In short, all the findings were negative. She was put on luminal two years ago and since then has had but one attack, and that two months after the initiation of treatment. She is now perfectly well and has given birth to another child since then. The optic discs still show atrophy.

TRAUMATIC EXTRAMEDULLARY HEMORRHAGE WITH SYMPTOMS DUE TO INVOLVEMENT OF THE LOWER CERVICAL ROOTS*

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This case is of chief interest to the surgeon, and belongs to that field in which accuracy of diagnosis is especially essential. A man receives an injury and shows signs referable to his nervous system. The question of an operation immediately arises. In the case about to be described, a correct diagnosis at the time of injury might have led to operative interference that would have saved the patient many months of helplessness.

The patient fell from a wagon, was unconscious for half an hour and paralyzed for five weeks. Then recovery began and the paralysis became localized in both upper extremities. The injury occurred in May, 1917, and the nature of the lesion was not suspected until three months later.

Patient: B. S.; 63 years of age; male; Hungarian; carpenter.

History. *Present Illness.*—The patient complains of pain in the upper extremities and weakness, especially of the left shoulder and forearm.

In May, 1917, the patient was standing up in a wagon which suddenly made a quick motion forward. He was thrown backwards to the ground which he struck with the back of his head and neck. For about a half hour he remained unconscious, and while in this state or immediately following it he was taken to his home in the country and put to bed. He was confined to bed for five weeks and says that he was unable to move. Whether this condition of prostration was due to pain or complete paralysis could not be ascertained, as the patient is senile and has great difficulty in answering questions directly. Several weeks later he came to New York and went to Bellevue Hospital, where he remained for three weeks. At this time an *x*-ray examination of his head and neck and a Wassermann reaction were negative. The patient states that during this period of his illness he was unable to dress or feed himself on account of the paralysis of his arm.

A short while after this the use of his arms began slowly to return, so that at present while there is an extreme limitation of motion, especially in his left arm, he is able in an awkward sort of a way to put on and take off his coat.

Concomitant with the motor symptoms were sensory changes. The patient said his hands and fingers felt as though they were frozen, and that later everything hurt him. About three months after the injury he was examined by Dr. Osnato who found an area of anesthesia in the interscapular region.

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* Presented at the Clinical Conference, December 31, 1917

Previous History.—A summary of the patient's previous history reveals no condition that has any bearing on the present illness. He can recollect no disease of childhood and says that he was perfectly normal up to the age of about twenty when he began to spit blood and was under the observation of Nothnagel, for tuberculosis. Patient drank in moderation, smoked excessively and had an attack of gonorrhea thirty-three years ago.

Fourteen years ago he fell from a step-ladder and received rather severe injuries in his arm and legs which incapacitated him for two months. In 1916, he was confined to bed for ten weeks with an attack of pneumonia. There is no history of dyspnea, kidney disease, convulsions, incontinence, headache, nor bowel difficulties. He has a slight cough. His wife had three children, and had no miscarriages. He has had no operations, nor hernia, nor evidence of any new growth. Syphilis is denied.

Family History.—The patient does not recollect his father, mother, or any other relative.

NEUROLOGICAL EXAMINATION

VOLUNTARY MOTOR SYSTEM. *Attitude.*—The left arm is held motionless and is not used in any unconscious way, such as making gestures.

Gait.—The gait is normal. The patient walks forwards, backwards, sidewise, and on the heels and toes in a normal manner.

Coördination.—There is no disturbance of coördination. The patient knows the positions of various parts of his body, and can properly direct those muscles which are not actually atrophic.

Skilled Acts.—There is no apraxia; imitation of skilled acts that the patient formerly knew can be performed without difficulty. Dyspraxia was present only in those acts which required the use of paralyzed or atrophic muscles. There was no dysarthria, anarthria, paraphasia, nor aphasia. There was some disturbance in the handwriting due to muscular weakness.

Reflexes (0 = absent, 1 = slight, 2 = active):

(*a*) *Deep:*

	Right	*Left*		*Right*	*Left*
Jaw-jerk	1	1	Ulnar	0	1
Pectoral	0	0	Wrist	1	1
Biceps	2	1	Suprapatellar	1	1
Triceps	1	0	Patellar	1	1
Radial	1	0	Achilles	1	1

	Right	*Left*
Periosteal	0	0

(*b*) *Superficial:*

	Right	*Left*
Ciliospinal	1	1
Supra-umbilical	1	1
Suprapubic	1	1
Upper lateral abdominal	1	1
Lower lateral abdominal	1	1
Cremasteric	1	1
Bulbocavernosal	Not taken	
Anal	Not taken	
Plantar flexion	1	1
Babinski	0	0
Chaddock	0	0
Oppenheim	0	0
Gordon	0	0
Schäfer	0	0
Fanning	0	0

Abnormal Involuntary Movements.—None present.

Muscle Strength.—Abduction of Shoulder: Right, normal; left, very slight movement.

Flexion of Forearm on Arm: Right, normal; left, normal.

Extension of Forearm: Right, normal; left, normal.

Flexion of Wrist: Right, normal; left, weak.

Extension of Wrist: Right, normal; left, weak.

Flexion of Fingers: Right, weak; left, very slight movement.

Extension of Fingers: Right, weak; left, very slight movement.

Opposition of Thumb: Right, normal; left, could not be performed.

Muscle Status.—Volume: Atrophy of the left supraspinatus and infraspinatus muscles.

Contour: Shows change in left supra- and infraspinatus.

Consistency: The left triceps and flexor muscles of left forearm are soft and flabby.

Tone: Atony in muscles indicated above.

Electrical Reactions: There was no reaction of degeneration although the responses were somewhat sluggish.

Irritability: Not tested.

Myoidema: Not tested.

Abnormal Associated Movements. — Not tested.

Summary of Voluntary Motor System.—There was definite evidence of a lower motor neurone type of paralysis in the muscles of the posterior scapular region on the left side, and suggestive evidence in the muscles of the left forearm of a previous paralysis of similar character. On the right side there was a certain amount of weakness that probably would have escaped unnoticed, were it not suggested by the condition on the opposite side.

NERVE STATUS.—Not examined.

GENERAL SENSORY. *Touch.*—Objectively, touch was diminished on the palmar surface of the left hand and fingers, lateral to the hypothenar eminence. Over this area, and extending up the lateral surface of the forearm and arm to about the midhumeral region, was an area of hyperesthesia. The back was generally hyperesthetic as far as the fourth cervical vertebra. At the interscapular region, no area of anesthesia was found.

Pain.—In this examination, the coöperation of the patient was poor. He complained of subjective muscular pain in the left arm, in the anterior humeral region.

Temperature and Muscle Sense.—Normal.

Summary of General Sensory.—The lack of coöperative ability and hysterical condition of the patient, made an accurate determination of his sensibility impossible. Yet there was definite evidence of a hyperesthesia existing approximately in the region indicated.

CRANIAL NERVES AND HEAD. *Olfactory and Nose.*—Normal.

Optic and Oculomotor Apparatus.—Normal.

Trigeminal and Mouth.—Normal.

Facial and Face.—Normal.

Acoustic and Ear.—Normal.

Glossopharyngeal, Vagal, Pharynx, and Larynx.—Normal.

Spinal Accessory.—Normal.

Hypoglossal and Tongue.—Normal.

MENTAL STATUS.—The patient endeavors to coöperate but is loquacious, and instead of answering questions directly, gives long details regarding irrelevant matters. His mental condition is characteristic of a mild grade of senility.

CRANIAL MORPHOLOGY.—Normal.

SYSTEMIC EXAMINATION. *Tegumentary.*—The nails of the left hand were a pearly white, and the skin of the finger-tips was purplish blue and glossy. The whole hand is cold.

Glandular.—Normal.

Cardiovascular.—Normal.

Respiratory.—Evidence of chronic bronchitis was present.

Gastro-intestinal.—Normal.

Genito-urinary.—There were no bladder disturbances at the time of the examination and the patient denied the existence of that condition at any time since the injury.

Skeletal.—Normal.

LABORATORY TESTS. *Wassermann.*—Negative.

Lumbar Puncture.—Not taken.

Blood Count.—Not taken.

Urine.—Negative.

X-ray.—Negative.

SUMMARY AND ANALYSIS

The progress of the case from the time of the injury up to the present falls into several stages:

First: The immediate stage of the injury itself, during which the patient became unconscious.

Second: A period of five weeks, during which no recorded observations were made. The patient may have been completely paralyzed, or he may have been hysterical, or he may have been unable to move on account of pain.

Third: The time during which the patient stayed in Bellevue Hospital under observation. A roentgenogram was made but showed no injury, and a blood examination was made with negative results. During the process of making a differential diagnosis, the patient became discontented and left the hospital. An injury to the brachial plexus was suggested.

Fourth: The last stage, which was the stage of recovery. The patient was able to dress himself and use his arms with greater freedom. Dr. Osnato observed a patch of anesthesia between the scapulæ which was of much importance, while the fact that this no longer exists shows a definite progress in repair.

What then have been the pathological processes that caused these phenomena, and where in the nervous system were they located? The paramount feature of the first stage was concussion of the brain, which masked the injury to the cord, so that in all probability the local, and more serious, injury was unrecognized. It is hard to conceive of any way in which a diagnosis of cord injury could have been made during this stage of unconsciousness.

In the second stage, however, there should have been many leading features, pointing to a localized lesion. A severe compression of the cord, or a hemorrhage into it, would have shown the signs of lower motor neurone involvement *at the level* of the lesion and upper motor neurone signs *below the injury*. Yet there is no record of this stage available. The patient says he could not walk or move, although he states that his bladder functions were normal. Nevertheless, in spite of the severity of this paralysis, he was able in five weeks to come to New York and seek advice at Bellevue Hospital. Whatever had previously transpired within the patient's nervous system had by this time begun to recede. That it was not a fracture was shown by the negative roentgenogram; that there was an actual severance of nerve tissue was shown by the rapid recovery. Some other tissue in close relationship with the nervous system must be sought as the seat of the lesion.

The nervous system is composed of ectodermal elements, the neurones and neuroglia, and mesodermal elements comprising the blood vessels and meninges. A sudden extravasation of serum into the meninges and nerve tissue itself would account for all the patient's symptoms up to the time of his recovery from the second stage.

The localized paralysis, which became more manifest as the acute symptoms passed off, indicated a more extensive damage than the simple exudation of serum and was very suggestive of hemorrhage. From the neurological standpoint, hemorrhages occur from two causes: (1) injury to the vessels themselves, or (2) vascular disease. And it is not an extremely unusual occurrence for a patient who has arterio-sclerosis to rupture a vessel as the result of an emotional stress caused by an accident. In the present case the arteriosclerotic lesion seems unlikely. The patient had no headaches nor any of those suggestive disorders that make one suspect arteriosclerosis, and moreover his blood pressure was only 110.

The nervous system is surrounded by unyielding membranes which allow no escape for any hemorrhage occurring within them, and consequently increased pressure caused by such a condition acts directly on the nerve tissue itself. Roughly speaking, the point of maximum pressure is at the center of the hemorrhage, and from this point it gradually diminishes in all directions until it reaches a pressure of zero at the periphery. The result of such different pressure on the nerve tissues varies in proportion from a simple pressure paralysis, to complete degeneration. As soon as a hemorrhage ceases, absorption and organization take place, and clinically there is a parallel recovery from pressure paralyses. If degeneration has taken place, another series of pathological changes develops which is quite independent of those processes which have to do with the absorption or organization of the hemorrhage. In the case under discussion, it would seem that the five weeks the patient was confined to bed corresponded to the absorption of the hemorrhage, and the course of the disease since then has corresponded to the pathogenesis of nerve degeneration itself.

If a nerve is severed, or cut off from its cell body, degeneration of the peripheral stump occurs. Centrally, degenerative processes usually take place in the fiber up to the nearest node or nodes of Ranvier and central chromatolysis in the cell body. If the point of injury is near the cell body, then the latter undergoes degenerative changes which may result in its final disappearance.

Granting that these pathological changes have occurred in this particular case, the next point is to establish the site of the lesion. A flaccid paralysis, with atrophy of muscles, means a damage either to the cells in the anterior horn of the cord, or to the nerves leading from them. If the paralysis is not accompanied by sensory changes then the lesion must be in the anterior horns; if sensory changes are present then the lesion involves either the posterior portion of the cord as well as the anterior, or the afferent nerves between the cord and the periphery. Peripheral nerves contain axones emerging from several segments of the cord, and injuries to them produce sensory phenomena, which correspond to the peripheral distribution of these nerves. A lesion of the roots, however, before they combine with roots from other segments to form the peripheral nerves, obviously has a radicular distribution. And as these roots give off a posterior branch before they enter into plexus formations a characteristic sign of their injury is anesthesia over the area of distribution of this posterior branch.

In the diagnosis of peripheral nerve injury it is necessary to find out, first, what nerve or level of the cord is damaged, and secondly, whereabouts in the course of this nerve the injury has occurred.

The motor symptoms definitely fix the level of the injury. Thus, in the present case the atrophy of the supra- and infraspinati, combined with the paralysis of the flexor muscles of the hand and fingers, would place the injury somewhere central to the upper trunk of the brachial plexus, and limit its level to the fifth, sixth, seventh, and eighth cervical and first dorsal segments. The bilateral sensory disturbance would at once centralize the lesion, and the area of anesthesia found by Dr. Osnato in the interscapular region rules out a double brachial plexus injury and places the lesion in the neighborhood of the spinal nerve roots.

The final step in the analysis is to locate the hemorrhage in its relation to the meninges. Beginning with the bony part of the vertebral canal, the spinal cord has the following coverings: (1) bone; (2) periosteum; (3) a space containing veins and fatty connective tissue; (4) dura mater; (5) the subdural space, containing lymph; (6) the arachnoid; (7) the subarachnoid space, containing cerebrospinal fluid; (8) the pia mater; (9) the cord.

The fact that the lesion was bilateral and concerned several segments of the cord would seem to rule out injury to the roots as they passed through the intervertebral foramina. The absence of any demonstrable changes in the bone or periosteum, as evidenced by the roentgenogram, would eliminate injury to the bone, periosteum, or space between these structures and the dura.

While hemorrhage into the cord substance itself must be seriously considered, it would be a risky diagnosis to make, in view of the fact that there were no physical signs below the level of the injury. A hemorrhage of the cord would, in the great majority of cases, show a change in the deep reflexes or in sensory phenomena referred to the lower part of the body.

A hemorrhage into the subarachnoid space would mix with the cerebrospinal fluid, gravitate to the lower extremity of the cord and produce symptoms in the legs. The subdural space, however, is normally merely a potential cavity. A hemorrhage into it would have to dissect its way up and down and the viscosity of the blood would prevent downward gravitation. Furthermore, this space extends around the cord and exudation into it would produce bilateral symptoms. It also contains the anterior and posterior roots of the spinal nerves, in a position extremely vulnerable to pressure. Therefore, the most logical diagnosis of the case under observation would be a subdural hemorrhage in the cervical region between the fifth and eighth segments.

DISCUSSION

Dr. O. S. Strong.—The findings in this case point to a root lesion. Of course one might theoretically get a double brachial lesion, but the picture points rather to radicular involvement, probably within the vertebral canal, the more exact localization depending upon the picture pointed out by Dr. Hoyt.

Dr. M. Osnato.—This man was hurt in a peculiar way. His head was jerked back as he fell, and he struck his back and the back of the head, becoming unconscious and paralyzed. The cord lesion was not recognized at first. The site of the injury to the cord is from the fifth to the eighth cervical. The first dorsal is not affected as the ciliospinal reflex is present. The total initial paralysis and subsequent gradual improvement points to hemorrhage. I do not think it was a root lesion, as it is unlikely that a hemorrhage would pick out the roots alone. The conception that I have is that we are dealing with a subpial hemorrhage.

DR. C. A. MCKENDREE.—We must bear in mind the fact that we are seeing the patient eight months after the injury, and in this period many symptoms have entirely disappeared. I think that the symptoms may represent one of two conditions: (1) 'commotion' of the cord with petechial hemorrhages from the fifth cervical to perhaps the first dorsal segment, or (2) residual manifestations of a hematomyelia localized in this region. The fact that pain sense is more involved than touch, while temperature sense is apparently normal, does not rule out the possibility that hematomyelia may have existed originally. We know from the clinical history obtained today that a great deal of restoration has taken place.

As to hemorrhage around the roots, the probability is that the blood would have gravitated lower down. I do not think we are dealing with a root lesion in this case.

DR. I. S. WECHSLER.—I must take issue with Dr. McKendree on the question of hematomyelia. All the signs seem to be against that, and the total absence of sensory dissociation I think fairly excludes such a lesion. Nor do I think that we are dealing with a peripheral lesion. As to the suggestion that a hemorrhage around the cord would have gravitated down the spinal canal and given distant symptoms, we can readily see how a small hemorrhage within the pia could be limited to a few sections of the cord. I think we are dealing with a radicular condition.

TWO CASES OF FRIEDREICH'S ATAXIA: BROTHER AND SISTER*

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These two cases came into the clinic looking and acting so much like a severe type of chorea that they might even have passed a hurried examination as such. The cases were presented at the first of the bimonthly conferences of the Neurological Department of Columbia University. I regret that there is no record of the discussion of these cases, the stenographer being absent on that occasion. The cases have special interest in showing the strong familial type of this disease. The two cases are very like one another in their history, development and symptomatology, and while there is no further evidence of the disease in the immediate family or ancestry (so far as I could ascertain), there are bad predisposing elements.

Case I: J. B.; No. 10537; age 12; male; Anglo-Saxon; United States; schoolboy.

HISTORY. *Present Illness.*—The boy complained of occasional headache without vomiting, of dizziness, and 'fidgety, shaking movements of the limbs'. The mother stated that she did not notice anything wrong with the boy up to the previous November (1916)—five months before his coming to the clinic, and she did not pay any special attention to his symptoms till about February, 1917. She first noticed unsteadiness in the gait and some 'peculiar' movements of the eyebrows. The headache complained of had lasted only for a few days, was associated with some slight dizziness (subjective) and there was also complaint of flatulent indigestion for the previous two months.

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* Presented at the Clinical Conference, July 12, 1917

Previous History.—Birth and early development normal; no trauma; no convulsions. Scarlet and measles at eight years of age.

Family History.—The family history is a little interesting. The mother is thirty-five; personal history negative. Four of her brothers and one sister died young from causes unknown. A second sister died at thirty, cause unknown. One brother of fifty is living, is epileptic, and has been in an institution for many years. Her father is a hard drinker, her mother died of cancer.

The father of the patient is forty-one and well, is not alcoholic, and has a negative personal history. One brother and three sisters are all well. There have been no nervous or mental diseases in the family. No information regarding the patient's paternal grandparents was obtainable and only a very incomplete account of the collateral branches of both families; but it was negative so far as it went.

VOLUNTARY MOTOR SYSTEM. *Attitudes.*—Nothing special except as noticed under other headings.

Gait (walking, slowly, fast; running).—The gait is unsteady; he comes down on the heels and has to balance more than normal. This is more marked with the eyes closed, when he staggers especially to the left. He cannot walk on the heels well, this bringing out the unsteadiness. Other tests bring out nothing additional.

Coördination:

(*a*) *Equilibratory.* With the eyes open there is some unsteadiness in standing with the feet together, the swaying being corrected continually by slight movements. With the eyes closed, staggering with a tendency to fall backwards (?). The patient cannot stand on the left foot alone either with the eyes open or closed. He can stand on the right foot a moment with the eyes open by an extreme balancing effort but not with the eyes closed.

(*b*) *Non-equilibratory.* The Finger-to-Nose and Finger-to-Finger Tests: These are performed imperfectly with waving movements both in and outside the plane of movement.

Pointing and Pass-Pointing: Give the same result.

Knee-Heel Test: Produces a similar result; irregular movements, variations in speed, and missing the knee. The right is in general better than the left.

Passing the Heel along the Shin: Shows the same irregular movements.

Toe to Finger held above: Wide side to side swaying right and left.

Dysmetria: Not marked.

Adiadochocinesis: Better with the right. Now and then some dysmetria, *e.g.*, slaps the chest.

Skilled Acts.—Speech: Although the speech was first noted as normal, apparently a slight disturbance can be detected; a slight unsteadiness in holding vowel sounds.

Writing: The letters are somewhat wavering but quite legible. The attempt to make a straight line shows some wavering and fine tremor.

Reflexes (1 = slight; 2 = active):

(*a*) *Deep:*

	Right	*Left*
Jaw-jerk	Absent	Absent
Pectoral	“	“
Biceps	“	“
Triceps	“	“
Wrist	“	“
Radial	“	“
Ulnar	“	“
Patellar	“	“
Suprapatellar	“	“
Achilles	“	“
Dorsocuboidal	“	“
Periosteal	“	“
Crossed periosteal	“	“
Ankle clonus	“	“
Patellar clonus	“	“

(*b*) *Superficial:*

Upper lateral abdominal	2	2
Lower lateral abdominal	2	2
Cremasteric	2+	2+

Plantar and Its Modifications:

Plantar flexion	1+	1
Babinski	Absent	Absent
Chaddock	“	“
Oppenheim	“	“
Gordon	“	“
Schäfer	“	“

Abnormal Involuntary Movements.—Tremors: General waving of the arms when extended in front of him. Tends to somewhat overextend the fingers and slightly flex the wrist.

Twitchings and Choreiform Movements: Noted in legs, trunk, arms, and fingers.

Athetoses, Spasms, Tics: None noted.

Muscle Strength.—Rising from Recumbent Position: Normal.

Flexion, Extension, Adduction, Abduction, Rotation at Various Joints: There is possibly a slight weakness in the terminal joint of the forefinger of the left hand and in flexion and dorsal extension of the left wrist. Elbows and shoulders normal. Possibly some weakness in plantar flexion of the small toes, both feet—more in the left, and also weakness of flexion at the left knee (?). Rotation, abduction, adduction, flexion, and extension of the thighs is normal.

Muscle Status.—No abnormalities noted.

Abnormal Associated Movements.—Noted under other headings.

NERVE STATUS.—Nothing noted.

GENERAL SENSORY. *Touch.*—Probably normal; if there is any diminution or alteration in touch discrimination it is in the toes of both feet.

Pain.—Apparently normal.

Temperature.—Normal, except possibly the feet.

Vibratory.—Absent in the terminal joint of the left big toe, both joints of the second toe, and the terminal joint of the left third toe.

Pressure.—Probably normal. If any diminution it is in the feet.

Posture.—Both feet in general good, except the toes which show some diminution. Also possibly some diminution in palmar flexion of the left wrist.

Stereognosis.—Probably normal. Certainly not absent.

Barognosis.—Nothing noted.

CRANIAL NERVES AND HEAD. *Olfactory and Nose.*—Not tested.

Optic and Oculomotor Apparatus.—Vision normal; discs normal; pupillary reflexes normal; no nystagmus. (Dr. Holden.)

Trigeminus and Mouth.—Normal.

Facial and Face.—Normal.

Acoustic and Ear.—Normal.

Glossopharyngeus, Vagus, Pharynx, Larynx.—Normal.

Spinal Accessory.—Normal.

Hypoglossus and Tongue.—Normal.

MENTAL STATUS.—Intellect is perfectly normal.

CRANIAL MORPHOLOGY.—Normal.

SYSTEMIC STATUS. *Tegumentary.*—Normal.

Glandular.—Normal.

Cardiovascular.—Normal.

Respiratory.—Normal.

Gastro-intestinal.—Normal.

Genito-urinary.—Normal.

Skeletal.—Pes cavus in both feet, of a marked type. Marked scoliosis, concave to the right. No distinct atrophy noted or asymmetry, except the scoliosis.

LABORATORY TESTS. *Wassermann.*—Negative.

Lumbar Puncture.—Not done.

Blood Count.—Not made.

Urine.—No report.

X-ray.—Not done.

DIFFERENTIAL DIAGNOSIS.—*See* summary at end of article.

Case II: E. B.; No. 10,538; age 14; Anglo-Saxon; United States; schoolgirl.

HISTORY. *Present Illness.*—Unsteady gait, uncertain (choreiform) movements of the limbs, twitching of the lips and fingers and 'rheumatic' pains in the legs. The mother noticed that the patient's back 'was not right' at the age of nine, but did not pay special attention to it. The other symptoms she did not notice until about two years previous to the patient's appearance at the clinic. These consisted in slight shaking movements of the head, unsteady gait, and trembling of the hands; she dropped things out of her hands. The mother thinks there is a possibility that the child was unsteady on her legs for a number of years.

Previous History.—Normal birth and development. Diphtheria at seven, whooping-cough at nine, frequent attacks of tonsillitis, measles at eight, and also a fall at seven or nine years of age with unconsciousness for 'ten minutes', but no subsequent effect.

Family History.—See Case I.

VOLUNTARY MOTOR SYSTEM. *Attitudes.*—No observations except as noted under other headings.

Gait (*walking, slowly, fast; running*).—The gait is unsteady, with some spreading, especially with the left leg. (Festination?) All this is more marked with the eyes closed. She comes down hard on her heels. Cannot walk on the heels. On stopping abruptly and on turning quickly she staggers. Walking a line she staggers to the left.

Coördination:

(*a*) *Equilibratory.*—She is unsteady with the eyes open and falls to the left, standing with the eyes closed (Romberg).

(*b*) *Non-equilibratory.*—Finger-to-Nose and Finger-to-Finger Tests: Performed with irregular movements and marked unsteadiness. The right is better than the left. Better with the eyes open.

Pointing and Pass-Pointing: She misses, passing usually to the outer side, especially with the left.

Knee-Heel Test: She misses the knee; the movements are irregular and poorer with the left.

Passing the Heel along the Shin: Wabbling.

Toe to Object above Foot: Somewhat poorer with the left.

Dysmetria: Nothing marked.

Adiadochocinesis: Not very marked.

Skilled Acts.—Speech: Appears to be normal.

Writing: Shaky, more unsteady than the brother's. The attempt to draw straight lines betrays fine tremor, about the same as in the boy.

Reflexes (0=absent; 1=slight; 2=active):

(*a*) *Deep:*

	Right	*Left*		*Right*	*Left*
Jaw-jerk	2	2	Ulnar	0	0
Pectoral	0	0	Patellar	0	0
Biceps	0	0	Suprapatellar	0	0
Triceps	0	0	Achilles	0	0
Wrist	0	0	Dorsocuboidal	1	0
Radial	0	0	Periosteal	0	0

	Right	*Left*
Ankle clonus	0	0

(*b*) *Superficial Reflexes:*	*Right*	*Left*
Upper lateral abdominal	2	2
Lower lateral abdominal	2	2
Plantar and Its Modifications:		
Plantar flexion	2—	2—
Babinski	Present	Present
Chaddock	0	0
Oppenheim	0	0
Gordon	0	0
Schäfer	0	0

Abnormal Involuntary Movements.—Tremors: None marked except what has been indicated in the writing.

Twitchings: Some fibrillary twitchings in front of the neck and chest.

Choreiform Movements: Practically in all parts, legs, trunk, arms, hands, neck, face (especially lips), jaw (?), and eyes (?).

Athetoses and Tics: None noted.

Muscle Strength.—Rising from Recumbent Position: Normal.

Flexion, Extension, Adduction, Abduction, Rotation at Various Joints: Plantar flexion of the toes possibly weak and extension of the terminal joints weak. In general the right is weaker than the left (patient is right-handed). Dorsal flexion of the foot limited right and left.

Muscle Status.—Nothing abnormal noted.

Abnormal Associated Movements.—No abnormalities except as noted under other headings.

NERVE STATUS.—Nothing abnormal.

GENERAL SENSORY. *Touch.*—Normal.

Pain.—Normal.

Temperature.—Not recorded.

Vibratory.—Probably normal, except the big toe joints in both feet.

Pressure.—Not recorded.

Posture.—Normal, except for slight disturbance, especially in the left big toe.

CRANIAL NERVES AND HEAD. *Olfactory and Nose.*—Not tested.

Optic and Oculomotor Apparatus.—Vision is normal, discs normal, reflexes normal, probably no nystagmus (possibly a slight degree looking to the left, at one observation).

Trigeminus and Mouth.—Normal.

Facial and Face.—Normal.

Acoustic and Ear.—Normal.

Glossopharyngeus, Vagus, Pharynx, Larynx.—No report.

Spinal Accessory.—No report.

Hypoglossus and Tongue.—Normal.

MENTAL STATUS.—Normal.

CRANIAL MORPHOLOGY.—Normal.

SYSTEMIC STATUS. *Tegumentary.*— Normal.

Glandular.—Normal.

Cardiovascular.—Normal.

Respiratory.—Normal.

Gastro-intestinal.—Normal.

Genito-urinary.—No report.

Skeletal.—Marked scoliosis, concave to the left. Slight stoop forward; marked pes cavus, more on the left.

LABORATORY TESTS. *Wassermann.*—Negative.

Lumbar puncture.—Not done.

Blood Count.—Not done.

Urine.—Not examined.

X-ray.—None made.

SUMMARY AND DIAGNOSIS

These two cases taken together show the gradual development of ataxia without remission, beginning at an early age—at least eleven in the case of the boy and nine to twelve in the girl, probably earlier in both. The gait is definitely ataxic in both, and there are marked generalized choreiform movements in both. Non-equilibratory coördination is much disturbed and about to the same degree in both. Speech is disturbed in one patient slightly, but the writing is affected in both cases. Practically all the deep reflexes are lost in both. The Babinski sign is positive in the girl. The sensory status is almost undisturbed in both, except for the changes in the toes. There is marked scoliosis and marked pes cavus in both children. The patients are brother and sister and there is a marked neurotic element in the family history. These symptoms, together with the familial history, make any other diagnosis than Friedreich's hereditary ataxia scarcely possible.

COMBINED DYSTROPHY WITH LOSS OF VESICAL CONTROL*

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The following case is of interest because it presents an unusual combined type of muscular dystrophy with the added rare feature of loss of bladder sphincter control.

Patient: John B.; age 34.

HISTORY. *Previous History.*—The patient came to the clinic on September 24, 1917, complaining of weakness of the muscles of the arms, leg, and back. The duration of the disease was eight or nine years, with a history of a progressive course. Venereal disease was denied. Six years ago the left kidney was removed following an injury. Alcohol has been indulged in only moderately. Tobacco has been used in very small amounts. He has had pleuropneumonia, an operation for mastoiditis, and a gland removed from the neck, otherwise the history was negative.

Family History.—The family history is of importance. A sister of the patient has the same kind of difficulty. She walks badly and is very weak, but not to the same extent as the patient. A brother has the same kind of muscular weakness and is getting worse steadily. There are no other members of the family, as far as is known who are suffering from any similar trouble.

Present Illness.—Up to eight or nine years ago, the patient was perfectly well except for the above mentioned illnesses. He noticed then a weakness of the back and thighs which later spread to the arms. It is difficult for him to describe the progress of the disease, except to state that he has grown steadily weaker up to the present.

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* Presented at the Clinical Conference, October 15, 1917

About eighteen months ago, he lost control of his bladder, and since that time has been compelled to wear a protective urinal constantly. There is absolute incontinence at the present time.

There has never been any pain, except for a dull aching in his legs after a hard day's work.

For several months his walking has been difficult, and especially so at night. When he starts to walk, he has a sensation of stiffness, but after a few minutes his muscles seem to 'loosen up'.

The bowels are regular and digestion is normal.

The patient has erectile power and is sure he is potent sexually. He has had eight or ten nocturnal emissions in the past six months.

PHYSICAL AND NEUROLOGICAL EXAMINATION

Voluntary Motor System. *Attitudes.*—As the patient stands, he leans upon his cane or upon a nearby object and has very evident muscle weakness, more or less generalized. In the sitting position nothing abnormal is observed.

Gait.—The gait is waddling in character, with the shoulders held back, the abdomen pushed forward, and the legs clumsily manipulated.

Coördination.—Coördination of the equilibratory and non-equilibratory types was normal.

Skilled Acts.—Skilled acts were normally performed except in those tests involving the paretic muscles.

Reflexes (1 = slight; 0 = absent):

(a) *Deep:*	*Right*		*Left*
Tricipital	1	=	1
Bicipital	1	=	1
Supinator	1	=	1
Patellar	1	=	1
Achilles	0	=	0

(The Achilles was found to be absent constantly with all forms of reenforcement.)

(b) *Superficial:*	*Right*		*Left*
Abdominals	1	=	1
Epigastrics	1	=	1
Cremasteric	1	=	1
Plantar	1	=	1

Abnormal Involuntary Movements.—There were no involuntary movements. Fibrillation in particular was not observed at any time.

Muscle Strength.—There was marked weakness of the deltoids and trapezii on both sides. The back muscle groups were very much diminished in power. The forearm and thigh groups on both sides were somewhat diminished in power. The manner of rising from the recumbent position was infantile in type.

Muscle Status.—The volume of the muscles, in general, varied markedly. The calf muscles were hypertrophic, in comparison with other groups. The shoulder and pelvic girdles showed marked atrophy. The consistency of the muscles varied also. In certain groups, especially those which showed atrophy there were decided flabbiness. In others, especially in the triceps and posterior tibial groups, there were areas of 'hard rubber' consistency. Tone was correspondingly affected.

The electrical reactions did not reveal a true reaction of degeneration. Faradic response was present throughout, the left side usually being greater than the right. To the galvanic current, the strength of the reaction varied greatly, but in all of the affected shoulder muscles, for example, the cathodal closing contraction was greater than the anodal.

Abnormal Associated Movements. — There were no abnormal associated movements.

Nerve Status.—Nothing abnormal was observed in the nerve status.

General Sensory.—The general sensory examination revealed no abnormality throughout to cotton wool, pin prick, and thermal tests.

Cranial Nerves and Head. *Olfactory and Nose.*—Smell was not tested.

Optic and Oculomotor Apparatus.—The fundi were negative. The pupils were equal, regular, central, and reacted to light and accommodation. Conjugate movements were well performed. There was no ptosis, strabismus, or nystagmus.

Trigeminal and Mouth.—The motor and sensory fifth were normal.

Facial and Face.—The facial movements were normal in all tests. Taste was not tried.

Acoustic and Ear.—Hearing was not tested. The vestibular tests were not applied because of the patient's weakness.

Glossopharyngeal, Vagal, Pharynx, and Larynx.—Swallowing was normal. Uvula rose in the midline. Voice was normal.

Spinal Accessory.—Nothing abnormal was discovered in the sternomastoids. The trapezii were markedly atrophied, especially in the upper portions.

Hypoglossal and Tongue.—The tongue protruded in the middle line and presented no atrophy and no tremor.

MENTAL STATUS.—The mental status was normal, considering the education of the patient.

CRANIAL MORPHOLOGY.—To inspection and palpation there was no abnormality in the skull.

SYSTEMIC. *Tegumentary.*—There was no abnormality of the skin or appendages.

Glandular.—The glandular status was normal.

Cardiovascular.—The cardiovascular system was entirely negative.

Respiratory.—The respiratory system was normal.

Gastro-intestinal.—There was no deviation from the normal in the digestive functions.

Genito-urinary.—Erectile power was preserved. There was absolute vesical incontinence.

Skeletal.—No abnormality was detected in the skeletal system.

LABORATORY TESTS. *Wassermann.*—The blood serum test for syphilis was negative.

Lumbar Puncture.—The spinal fluid was negative for syphilis and presented no other abnormal features.

Blood Count.—A differential blood count was not made.

Urine.—Analysis was not made.

X-ray.—*X*-ray was not made in this case.

GENERAL SUMMARY

In summing up the abnormal features of this case I would emphasize the Aran-Duchenne type of shoulder-pelvic girdle atrophy; the Erb's type of pseudohypertrophic dystrophy, the absent ankle jerks, and the vesical incontinence. All of these occur in a man who is one of three who are similarly affected (except for the bladder symptoms). This case, though not typical of any one type of dystrophy, apparently represents a combination of three well-known types of myopathy.

DISCUSSION

DR. OSNATO.—I think that there have been some cases of mixed myopathies described. There is no reason why the several types, even the neurotic, combined with the myopathic, should not occur in the same individual. This case seems to be an example of three types: first, the hypertropic type of Duchenne; second, the spinal neurotic type; and third, the juvenile type of Erb.

STUDIES ON THE MORPHOLOGY OF THE GRAY MATTER IN THE BRAIN STEM OF VERTEBRATES BY THE RECONSTRUCTION METHOD

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PRIMATES

THE GRACILE AND CUNEATE NUCLEI IN THE GORILLA (STUDY NO. 1)

In neurology perhaps more than any other department of medicine comparative anatomy plays a predominant rôle. There are many structures in the human nervous system that would have remained in obscurity had not the study of the lower forms of life unmasked their real significance. The independent function of the spinal cord of vertebrates, for example, is lost during the process of evolution, and eventually becomes subservient to higher coördinative centers. The olfactory lobes, the optic thalamus, the corpora quadrigemina, the cerebellum, pons, etc., are also relatively of greater importance in some species than in others. Thus, in order systematically to present these variations I have undertaken a study of the entire gray matter in the brain stem of vertebrates, by the reconstruction method, with the idea in view of casting light on those structures that are now so problematic in man. The present series consists of the following primates: Homo sapiens, Troglodytes gorilla, Troglodytes niger, Simia satyrus, Hylobates syndactylus, Cynocephalus babuin, Macacus rhesus, and Lemur Cotta.

A serial section of the brain stem of each specimen is cut and stained by the Weigert-Pal method. The sections are fifty micromillimeters in thickness. Every fifth section is projected upon paper at a magnification of seven and a half diameters, and an outline of each nuclear (gray) structure is drawn and transferred to wax (one and a half millimeters in thickness). The wax is then cut, piled and modelled according to the Born method of reconstruction. On account of an unusually clear definition in the sections of the gorilla series, this specimen is the one I have chosen as the subject of my first description.

Although this study was undertaken as an anatomical contribution, its clinical utility is obvious. The three dimensions of a reconstruction emphasize relationships in a manner that the mind cannot grasp by the cross-section method of study; and as neuroanatomy and clinical neurology are mutually dependent, it is hoped that this report will give a clearer conception of anatomical relationships, which will be of assistance in diagnosis.

In order to bring this study to the attention of the clinical neurologist, I have received the permission of the editorial board of the Neurological Bulletin to publish the results of this work as a series of short descriptive articles.

Each article will have as a theme some particular nucleus (or group of nuclei), the description of which will be prefaced by a brief survey of the system to which it belongs, and although it is not within the province of this report to enter into diagnostic problems or clinical discussions, mention of diseases or syndromes will occasionally be made in order to illustrate an anatomical point.

The graphic method of description will be used as far as possible and will conform to the following system:

1. Selected drawings of cross-sections from which the reconstruction was made. In these illustrations, only the outline of the periphery of the brain-stem and nuclear structures is indicated.
2. Photographs of that portion of the reconstruction under discussion. (These are not supplied for Study No. 1.)
3. Schematic representations of the above photographs.

The present description will deal with the first "relay station" of the afferent pathway that carries, combines, and distributes deep sensibilities and tactile discrimination. Clinically, interruption of the *first neurone* of this sensory system gives the familiar picture of tabetic dissociation. Symptoms referable to the *second neurone* are usually caused by syringobulbia; and to the *third neurone* by a vascular disease or new growth in the thalamus, or postcentral convolution. The *second neurone* may be involved in a hemorrhagic lesion of the medulla, pons, or crura that includes other systems as well.

The cell bodies of neurones located in the posterior root ganglia give off processes which immediately bifurcate. One bifurcation is peripheral and terminates in an end organ, the other is central and passes into the posterior column of the cord, where another bifurcation occurs—one branch of which passes cephalad, the other caudad. The cephalic branch either passes into the gray matter of the cord or upwards by way of the posterior columns to the medulla. The posterior columns have two divisions, the column of Goll and the column of Burdach, and consist, in the main, of the ascending arms of the central bifurcations of the central processes of posterior root ganglion cells. These are the afferent *primary* neurones that convey muscle, joint, and tendon senses, the sense of vibration, and tactile discrimination.

The column of Goll is mesial and carries impulses from the legs and lower trunk, the column of Burdach is lateral and conveys afferent impulses from the upper trunk and arms. The axones which form these columns terminate in the nuclei of the columns of Goll and Burdach which are recognized on the dorsal aspect of the medulla as the clava and cuneus respectively. Axones arising from the cell bodies in these nuclei form the *second neurone system* and pass by way of the internal arcuate fibers and median fillet to the thalamus. From the thalamus the *third neurone* proceeds to the postcentral convolution of the cerebral cortex by way of the thalamocortical radiation.

A cross-section of the lower portion of the pyramidal decussation (Fig. 1) shows the remains of the anterior horns of the cord, above which is a narrow strip of reticular formation. Passing above the reticular formation is a groove formed by the pyramidal tracts, as they migrate laterad to take up their characteristic position in the cord. Dorsal to the pyramidal groove is the continuation of the posterior gray horn including the gelatinous substance of Rolando, which in this region is the nucleus of the descending sensory root of the fifth cranial nerve. The central portion of the figure is occupied by the central gray matter.

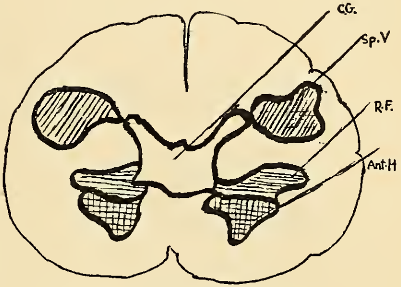

Fig. 1. (Sec. 145)

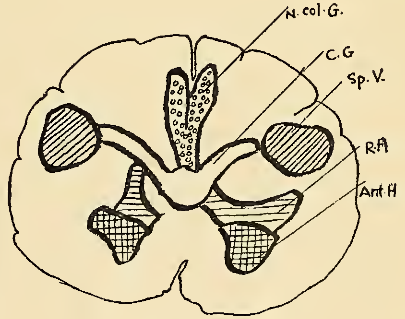

Fig. 2. (Sec. 150)

Fig. 2 is 250 micromillimeters cephalad of Fig. 1 and shows the presence of a new gray structure, lying in the posterior columns—the nucleus of the column of Goll. A groove has appeared between the central gray matter and the reticular formation occupied by the axones of the cells of the nucleus of the column of Goll, as they pass ventrally and mesially to form the median fillet.

The following abbreviations are used in the illustrations:

Ac. Inf. O.	Accessory Nucleus of Inferior Olive
Ant. H.	Anterior Horn
Br.	Bridge between Sp. V. and N. Col. B.
C. G.	Central Gray Matter
Col. B.	Column of Burdach
Col. G.	Column of Goll
G. G. C.	Gasserian Ganglion Cell Body
Inf. O.	Inferior Olive
M. F.	Median Fillet
N. Col. B.	Nucleus of Column of Burdach
N. Col. G.	Nucleus of Column of Goll
N. tr. vest.	Beginning of Vestibular Complex
P. R. G. C.	Posterior Root Ganglion Cell Body
Pyr. T.	Pyramidal Tract
Pyr. G.	Pyramidal Groove
R. F.	Reticular Formation
Sp. V.	Descending Sensory root of Trigeminus

Still another 250 micromillimeters cephalad (Fig. 3) another gray structure is found on the lateral arms of the central gray matter. It is triangular in shape and is the first appearance of the nucleus of the column of Burdach.

From this point cephalad, a gradual enlargement of these nuclei takes place, until they present the appearance shown in Fig. 4 (5mm. cephalad of Fig. 3). The expansion is not always confined to a mere enlargement of the nuclei as first observed, for in some sections one will note that a region of the posterior columns becomes suddenly studded with gray masses. On tracing these masses cephalad, they are seen to coalesce and unite with the nuclei indicated in the drawings. With a magnification of 7.5 diameters, it is impractical to attempt a reconstruction of such minute details.

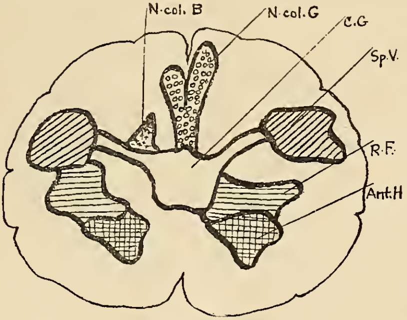

Fig. 3. (Sec. 155)

In Fig. 4 (5.25 mm. cephalad of Fig. 3) it will be noted that the nucleus gracilis has blended with the nucleus cuneatus, and that the latter is connected to the descending root of the trigeminus by small bridges. In the human, these bridges form the roof of a tunnel, which is bounded *laterally* by the descending root of the trigeminus, *ventrally* by the reticular formation and mesially by the nucleus of the column of Burdach; the tunnel terminates blindly by the gradual approximation of the spinal root of the trigeminus and the nucleus of the column of Burdach. This approximation in the gorilla occurs so shortly after the bridges have appeared that under a low magnification no tunnel is formed. In passing, attention should be called to the disappearance of the anterior horn, and of the pyramidal groove.

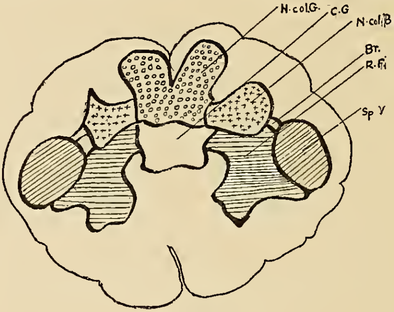

Fig. 4. (Sec. 260)

The proprioceptive nature of the spinal root of the fifth nerve is still a matter of debate, but the intermingling of its substance with the nucleus of the column of Burdach would seem strongly to suggest a proprioceptive function.

The function of the central gray matter is unknown, and that of the reticular formation is said to be intersegmental association. The remainder of the gray matter in this region of the brain almost entirely represents the great proprioceptive relay station of the entire body, and in it, from *within outwards*, are represented the lower limbs, trunk, upper limbs, neck, head and face.

The furrow between the gracile nuclei deepens (Fig. 5—2.75 mm. cephalad of Fig. 4), and at the same time a dorsal projection appears upon the central gray matter. Eventually those structures meet, and the dorsal projection splits as far as the central canal, so that a complete separation of the gracile nuclei occurs. The floor of this furrow then becomes the floor of the fourth ventricle (Fig. 6—250 micromillimeters cephalad of Fig. 5).

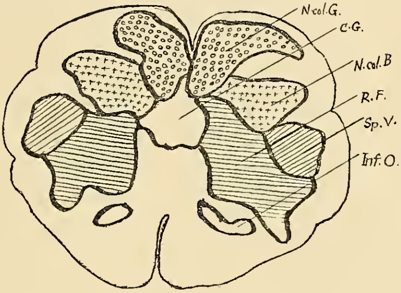

Fig. 5. (Sec. 315)

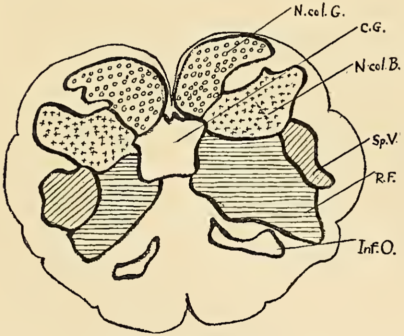

Fig. 6. (Sec. 320)

After this change has occurred, the walls of the fourth ventricle rapidly diverge, the floor extending dorsally, over the nuclei of Goll. These nuclei, and the nuclei of the columns of Burdach are deflected outwards and, on account of the dorsal extension of the ventricular floor, then occupy a lateral instead of a dorsal position.

Fig. 7 (3.25 mm. cephalad of Fig. 6). The floor of the fourth ventricle has opened and assumed its characteristic form. The nuclei of the columns of Goll have almost disappeared and the nuclei of the columns of Burdach have become greatly diminished in size. New proprioceptive nuclei—of the vestibular nerve—have begun to make their appearance. In Fig. 8 (dorsal view) I have endeavored to represent diagrammatically a cross-section of a reconstruction of the brain stem of the gorilla as it would appear if all the white matter were dissected away. Fig. 9 shows the lateral view of the same portion.

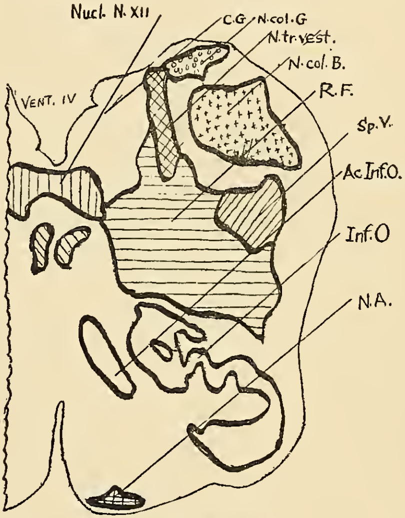

Fig. 7. (Sec. 385)

Turning back to Fig. 1, the gray matter will be seen to consist of four components: (1) the central gray substance, (2) the spinal root of the fifth nerve, (3) the reticular formation, (4) the anterior horns. The dorsal outline of this figure passes over the nucleus of the trigeminus of one side, then over the central gray

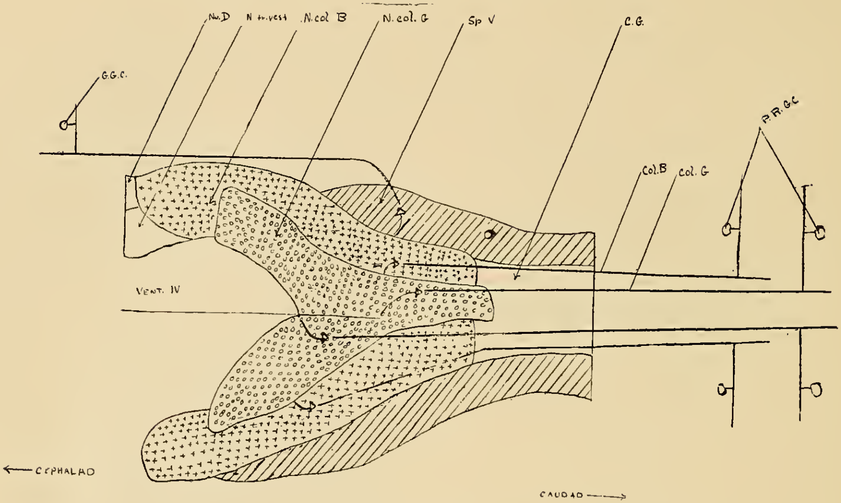

Fig. 8. Diagrammatic Representation of Reconstruction of Brain Stem of the Gorilla, Showing Form and Relations of Nuclei of Columns of Goll and Burdach. Dorsal Aspect

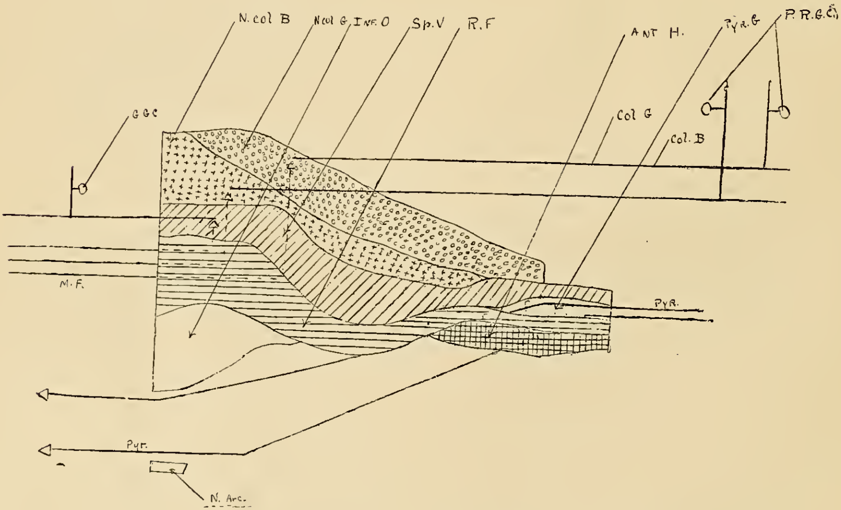

Fig. 9. Diagrammatic Representation of Reconstruction of Brain Stem of the Gorilla, Showing Form and Relations of Nuclei of Columns of Goll and Burdach. Lateral Aspect

matter, and then over the trigeminus nucleus of the opposite side. The lateral outline from above downwards passes over the nucleus of the trigeminus at which point it is interrupted by a groove. It then reappears as the lateral outline of the reticular formation and passes downwards and mesially over the anterior horns.

The examination of Figs. 2, 3, 4, 5, and 6 shows the dorsal outline has become changed, due to the presence of the nuclei of the columns of Goll and Burdach, and that the lateral outline has become modified by the presence of these same nuclei, by the disappearance of the anterior horns, and the pyramidal groove. The beginning of the inferior olivary nucleus is seen from this view. Fig. 8 shows the form of the gracile and cuneate nuclei, their relations to one another and to the spinal root of the trigeminus; Fig. 9 gives their shape and relations from the lateral aspect.

In Fig. 7 the dorsal outline covers the floor of the fourth ventricle, the dorsal triangular nucleus and nucleus of the descending root of the vestibular nerve, the remnant of the nucleus of Goll and the cuneate nucleus, while the lateral outline includes the same structures as in the preceding figures, with the exception of the nucleus arciformis which has now appeared. The dorsal surface of the reconstruction shows that as the fourth ventricle appears a divergence of the gracile nuclei occurs, and that the effect of this lateral extension is to push the cuneate nuclei and the nuclei of the trigeminus outwards.

The close relationship of the cuneate nucleus to the nucleus of the vestibular nerve as shown in Fig. 8 seems quite obvious when one considers that the nucleus cuneatus is proprioceptive on account of its position in the muscle sense system, and that Deiters's nucleus and the nuclei of the vestibular nerve are also proprioceptive on account of their connection with the semicircular canals.

The next article will be a description of the nuclei of the vestibular apparatus at which time a summary and conclusion in regard to the proprioceptive system will be made.

A CASE OF MULTIPLE SCLEROSIS WITH KYMOGRAPHIC STUDIES OF THE TREMOR*

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Multiple sclerosis has been so frequently mistaken for hysteria, even by able clinicians, that the following case will be of interest from that point of view.

Patient: E. D.; No. 13,031; age 37; female; Anglo-Saxon; United States; housewife; married.

HISTORY. *Present Illness.*—This patient came to the clinic September 27, 1917, complaining of what she called "shaking of the arms" and difficulty in speaking. She also spoke of "fainting spells," which were infrequent, and of unsteadiness in walking.

The onset was apparently gradual, beginning, according to her first statement, three years ago. She first noticed that she could not talk well. She does not remember whether her arms began to shake at that time but she is sure that this symptom was present soon after. Shaking movements of the head were also noted by her at about the same time. She thinks the symptoms came on as the result of childbirth (January 28, 1912). She was hysterical, she says, before the baby was born, and she remembers the shaking of the head at that time. This would make the onset nearer five years ago. She states that, previous to this time, she never had "hysterical attacks." The shaking movements have been getting steadily worse since the onset. They have never entirely disappeared but there have been periods of diminution in intensity. It is the same with the speech difficulty. All symptoms are worse at the time of the menses. She states that emotional excitement does not aggravate the symptoms. The tremors all cease on lying down. She does not complain of pain.

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* Presented at the Clinical Conference, June 12, 1917

There is evidence that there has been occasion for emotional outbreaks of an hysterical character. She speaks of unhappy domestic relations, with considerable complications in and outside of the home. The patient states that she was "terribly nervous" during those experiences.

Two years ago the right leg occasionally gave way suddenly. This happened twice in the street, when she fell to the ground. She has had about half a dozen such attacks in all, most of them at home.

The first fainting attack occurred when her baby was six months old, and the last one was last winter—an interval of about five years. There may have been six such attacks in all. From the patient's description they appear to have been attacks of true syncope. There were no convulsions at any time.

Previous History.—The patient says her own infancy and childhood were normal as far as she knows. She did not have convulsions, chorea, or other nervous manifestations. She is said to have had Bright's disease at seventeen and rheumatism at eighteen. Her school life seems to have been normal.

Family History.—Father is fifty-five and alcoholic. Mother died at forty-seven, and was epileptic. No information could be obtained concerning paternal and maternal grandparents. The history of uncles, aunts, and collateral relatives was negative.

VOLUNTARY MOTOR SYSTEM. *Attitudes.*—Nothing abnormal is noted, except as appears under other headings.

Gait.—The gait is unsteady and irregular, with tremulous jerkings. The various forms of testing the gait bring out the same results and nothing additional. The gait is not spastic. The patient does not come down on the heels nor throw back the knee.

Coördination:

(*a*) *Equilibratory.*—On endeavoring to stand with the feet together there is varying marked swaying and irregular movements of the whole body. The patient is very unsteady and loses her balance. All this is increased with the eyes closed. She cannot stand on either foot alone. Both sides are similarly affected.

(*b*) *Non-equilibratory.*—Finger-to-Nose Test: There is a coarse tremor with wide excursions aggravated as the finger is brought in proximity to the nose, involving the arms and shoulders. The tremor is more marked on the left than on the right.

Finger-to-Finger Test: Brought out the same result.

Pointing and Pass-Pointing: Bring out the same tremors, the left arm being more affected than the right.

Heel-to-Knee Test: There are unsteady wabbling movements, about equal on both sides.

Heel-to-Knee along Shin: This test brings out the same ataxic movements on both sides.

Toe-to-Finger held above: She does this test fairly well, but with considerable unsteadiness on both sides.

Skilled Acts.—Speech is jerky, slow, with apparent dysphonia. It is not certain whether the difficulty is articulatory. The speech is hard to describe. It seems to me that it does not come under the ordinary conception of scanning. The difficulty seems to be with phonation and with the muscles governing respiration. Attempts at writing reveal that she is unable even to make a letter. She holds a pencil in both hands to steady it; the result is only a few irregular marks.

Reflexes (1 = slight, 2 = active):

(*a*) *Deep:*

	Right		*Left*
Jaw-jerk: Not obtained.			
Pectoral	1	=	1
Biceps	2	=	2
Triceps	1	=	1
Patellar	1	=	1

The patellar reflex is difficult to get at times even on reinforcement.

Achilles	1	=	1

The same difficulty as with the patellar.

Dorsocuboidal and periosteal: Not obtained.

Ankle clonus: Not present R = L
Patellar clonus: Not present R = L

(*b*) *Superficial:*

Upper lateral abdominal R = L

At another time the left was not obtained.

Lower lateral abdominal R = L

Both slight. At another examination neither of these was obtainable.

Plantar: A Babinski reflex was not obtained on either side upon repeated examinations; but today a doubtful Babinski appeared on the right side.

Gordon and Schäfer: These were definite on the right side, but were not present on the left.

Oppenheim: Not obtained.

Gordon, Schäfer, Babinski: Today neither the Gordon nor Schäfer were obtainable, while, as has been said, the Babinski appeared.

Abnormal Involuntary Movements.—Tremors: Coarse tremors were especially noticeable in the arms and hands and included the shoulders. The tremor is both motofacient and non-motofacient. It is increased markedly by the finger-to-nose and other tests already mentioned. It disappears on complete relaxation. There is a mild negation tremor of the head, which also ceases on lying down.

Twitchings, Athetoses, Spasms, Convulsions: None.

Muscle Strength.—No paresis or paralysis of any of the muscle groups.

Rising from sitting position was normal except for irregular muscular movements already noted.

Rising from recumbent position was performed in the same manner.

Flexion, extension, adduction, abduction, and rotation at various joints showed the muscle strength to be normal and equal on the two sides.

Muscle Status.—Muscle tonus is normal.

Abnormal Associated Movements.—Not examined specifically.

Summary and Interpretation.—In the examination of the motor system as recorded here, we find marked ataxic gait, marked ataxia in the coördinated movements of the upper and lower limbs, a difficulty in speech, which might be called ataxic phonation, and absolute inability to write, owing to the wide excursions of the tremor. The deep reflexes are all present but in the case of the patellar and Achilles reflexes they were sometimes difficult to elicit.

There was positively no exaggeration of any of the reflexes at any time. Of the superficial reflexes, the abdominals are very slight and the left upper abdominal is more doubtful than the right. The Gordon and Schäfer reflexes, however, are present on the right side but do not seem to be constant either as they were not obtainable at the last examination. A doubtful Babinski is also referred to this side. On the left these tests were negative. There is no sign of spasticity or hypertonicity, and the muscle strength is normal and equal throughout.

NERVE STATUS.—No abnormalities.

GENERAL SENSORY. *Touch.* — Normal.

Pain.—While she occasionally does not seem to recognize the pin-point as such (calls it dull) this is not constant, and repeated tests over the same area on other occasions were normal (same for trunk, legs, and arms).

Temperature, Vibratory, Pressure, Muscle-tendon, and Stereognosis.—Normal.

Barognosis.—Not tested.

CRANIAL NERVES AND HEAD. *Olfactory and Nose.*—Not tested.

Optic and Oculomotor Apparatus.—Normal. Excessive concentric contraction of the fields (functional). Eyes otherwise normal. The fundi are normal and there is no sign of nystagmus. Light and accommodation reflexes are normal. (Dr. Ward A. Holden.)

Trigeminal and Mouth.—Normal.

Facial and Face.—Normal.

Acoustic and Ear.—Normal.

Glossopharyngeal, Vagal, Pharynx, Larynx.—Uvula rises normally. Pharynx and pharyngeal reflexes are normal; there is no difficulty in swallowing.

Spinal Accessory.—Normal.

Hypoglossal and Tongue.—Tongue movements are normal.

Summary and Interpretation.—The findings of the above tests are negative. Nystagmus and pallor of the optic discs are absent.

MENTAL STATUS.—The patient is perfectly normal intellectually. Responses are prompt and intelligent; coöperation is good. Emotional instability is very apparent; she is easily made to laugh in an exaggerated way. I have not observed any tendency to depression or hysterical weeping.

CRANIAL MORPHOLOGY.—Not investigated.

SYSTEMIC STATUS. *Tegumentary.*—Normal.

Glandular.—Thyroid gland normal; no evidence or suspicion of any other glandular disturbance.

Cardiovascular.—Heart and vessels normal.

Respiratory.—Lungs normal.

Gastro-intestinal.—Nothing abnormal.

Genito-urinary.—Menstruation is normal. The patient states that on two occasions there was involuntary dribbling of urine since her first appearance at the clinic. On both occasions the loss was very slight.

Skeletal.—Normal.

Summary and Interpretation. — There is nothing of importance here unless the slight urinary disturbance is to be considered.

LABORATORY TESTS. *Wassermann.*—Blood: November 1, 1917, negative.

Lumbar Puncture.—Not done.

Blood Count.—Not done.

Urine.—Normal.

X-ray.—Not taken.

SUMMARY

Here we have a definite ataxia, equilibratory and non-equilibratory. The patient cannot stand steady with the feet together and cannot stand at all with the eyes closed; she has an ataxic gait, a coarse intention tremor of both arms and legs, with a mild tremor of the head. She has a dysphonia; also an absolute inability to write, owing to the wide excursions of the tremor. There is a strong probability of Gordon and Schäfer on the right side, practically certain, and the possibility of a Babinski on that side (doubtful). The upper lateral abdominals are possibly unequal, the left less constant than the right. The patient gives a history of gradual onset over a period of five years, including several spells of falling, due to a sudden giving way of the right leg; no loss of consciousness. She also gives a history of several (probably six) fainting spells, the cause of which is not made clear and, further, several attacks of an "hysterical" nature. These symptoms point rather definitely to a diagnosis of multiple sclerosis although the reflexes are nowhere exaggerated and are even diminished in the legs, and there is no sign of spasticity; and there is an absence of fundus changes and nystagmus. The tracings of the tremor made by Mr. De Bra are of the multiple sclerosis type, as will be seen in the accompanying illustration.

Against the diagnosis of multiple sclerosis might be considered: the lack of spasticity in the gait, the diminished deep reflexes, the absence of any muscular rigidity, the absence of any motor weakness; also that there is no nystagmus and no changes in the fundi, as well as the fact that the intellect is clear; there is no mental dullness or deterioration. Finally, the gradual onset and progression of the disease instead of periods of remission might be slightly against the diagnosis. The positive findings, however, are too definite and pronounced to be outweighed by any of the negative ones mentioned, and the possibility of cerebellar disease or myelomalacia is very remote. A "pseudosclerosis which cannot be distinguished from a real multiple sclerosis" is spoken of in the literature (Westphal). I do not know whether a functional type of this disease is meant by this or not, so I merely mention it.

DISCUSSION

MR. DE BRA.—There was some doubt about the clinical diagnosis of this case; it seemed like a multiple sclerosis, yet there were elements that led to the suspicion that it might be functional. To settle the matter in a way that would seem to clinch it, the patient was sent over to the laboratory and this graph (Fig. 1) made.

The procedure is as follows: From a group of muscles we isolate two units that are synergic when there is no organic disturbance

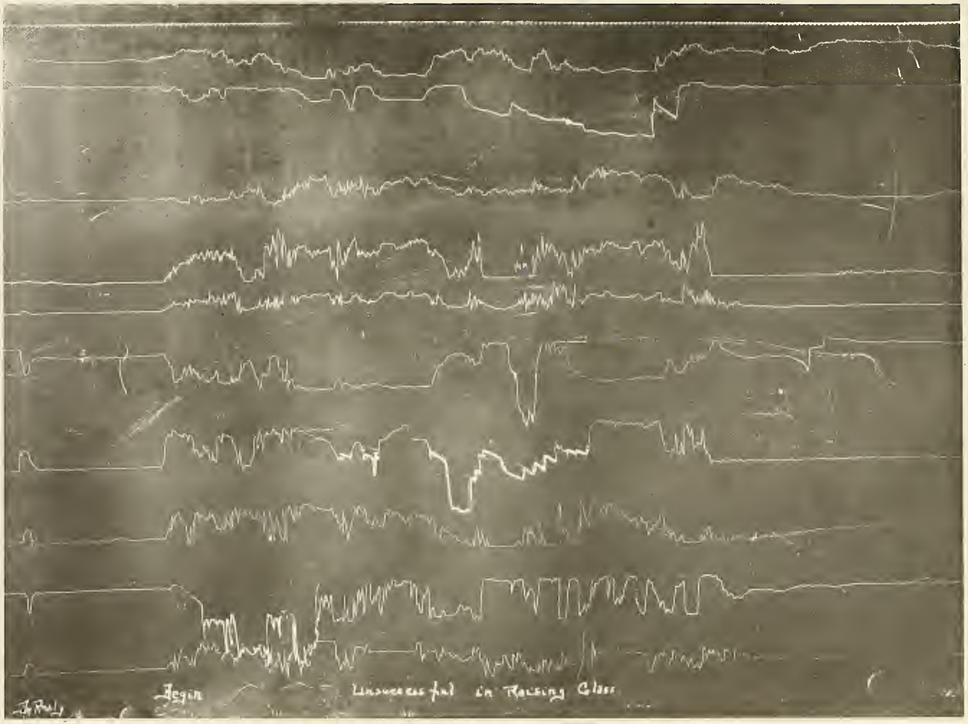

Fig. 1. Kymographic Record Showing Typical Curve of Multiple Sclerosis

Records taken of six muscle groups and three different motion curves

to prevent it. In the motor disturbances that are called tremors there is a definite relation between two muscle groups, such as that between the flexors and extensors of the wrist, for instance. When that synergic relation is not present, as in multiple sclerosis, it is not a true tremor but an ataxia. In functional cases we can trace on the graph a synergic pattern, which speaks for the diagnosis; for in these functional tremors we find the normal relation between the antagonistic muscles perfectly maintained, which in multiple sclerosis is broken, the unit groups of the antagonistic muscles showing no synergy whatsoever. A glance at the graph here presented shows the asynergy of the muscle units in this case, the tracing being perfectly characteristic of true multiple sclerosis.

Dr. Frederick Tilney.—I think the important point in regard to these graphs is that we can see the fundamental disturbance in muscle group of the synergic units. This synergy is lost in multiple sclerosis, the muscles being innervated more or less independently, which is true in ataxia rather than tremor. It, therefore, seems better to speak of the gross motor disturbance in multiple sclerosis as profound dystaxia rather than tremor.

Dr. W. A. Holden.—It is rather extraordinary not to find nystagmus when there is such marked ataxia in other parts of the body, but occasionally in multiple sclerosis one finds almost total absence of nystagmoid movements. Usually it can be brought out by persistent tests, but in this case there is entire absence of true nystagmus. However, this does not militate against the diagnosis.

Dr. M. Osnato.—I think one point ought to be brought out in the discussion, and that is the extreme difficulty of differentiating between hysteria and multiple sclerosis at times. Some of the mistakes are made because the fact is lost sight of that in multiple sclerosis there are changes in the emotional sphere and later in the intellectual. This case illustrates this point; the patient is in a frame of mind where she laughs easily and seems to be singularly free from any insight into the importance of her physical condition. In the absence of nystagmus, fundus findings, and scanning speech—though the last is a factor in this case—one is apt to be thrown off the track and the diagnosis made difficult. This case seems to be a cerebellar type of the disease; at least the scanning speech, incoordination and ataxia would lead one to that conclusion, together with the absence of marked spasticity and the comparatively diminished reflexes.

Dr. O. S. Strong.—I have nothing special to say in regard to this case except to call attention to what is obvious—the non-typical character of the reflexes for multiple sclerosis, and also the absence of spasticity. While we all agree that the ataxia is probably cerebellar, yet it seems to me the abnormality of movement does not conform exactly to that seen in cerebellar lesions.

Dr. Charles A. McKendree.—To my mind there is no doubt of the correctness of the diagnosis of this case, and it emphasizes what I believe to be a fact: namely, that you can diagnose multiple sclerosis without the presence of any of the classical signs or symptoms.

Only recently I had a case which would illustrate this point. A trained nurse of an exceedingly nervous type came to me, complaining of weakness in the arm and leg of the left side. As she had always been of an introspective and apprehensive nature and because no gross symptoms were present, she was sent to me as a case of hysteria. Examination, however, revealed bilateral Babinski and temporal pallor of one disc. In the absence of all other organic signs I made the diagnosis of multiple sclerosis, and I am sure that the progress of the disease will confirm the diagnosis. Multiple sclerosis is a disease which presents a multiplicity of symptoms and signs, none of them necessarily conforming to the classical triad of Charcot.

The only other point that occurs to me is a practical question. Here is a woman at the child-bearing period of life. If she should become pregnant, what should be the attitude of the physician towards her pregnancy as regards the progress of her disease? We know, of course, that many cases of multiple sclerosis may go through a normal pregnancy, but I would like to know the opinion of the Conference upon this point.

Dr. L. S. Aronson.—I have had two occasions to deliver women suffering from multiple sclerosis, the first being eight years ago. The patient's condition was not aggravated by gestation, delivery, or the puerperium. The second case was seen four years ago; she had no bulbar symptoms prior to her pregnancy, but after delivery developed bulbar signs rapidly and died within a few months. In both cases the offspring was normal.

Dr. Frederick Tilney.—I should like to add to Dr. McKendree's discussion without answering his sociologic question. Friedrich Müller showed that in twenty-five per cent. of the cases of multiple sclerosis there is absence of nystagmus, of scanning speech, and even of intentional tremor. The term *multiple sclerosis* is too much limited in this country; we are still under the spell of Charcot's teachings and demand that his triad be present before we are willing to make a diagnosis. Multiple sclerosis makes itself manifest in so many ways that we cannot properly adhere to that older conception. To my mind, the absence of any of the cardinal symptoms is not against the diagnosis; in fact, I believe that often lateral spinal sclerosis and even some of the lower motor neurone diseases should be diagnosed as multiple sclerosis.

Dr. Hallock.—In reply to Dr. McKendree's question, I should be inclined to leave the matter to the patient, should such a contingency arise. If one were sure she could endure it and she wished to have a child, I should not interfere.

This case interested me because the literature lays such stress on the cardinal symptoms of this disease, as Dr. Tilney has pointed out, and here is a case in which several of them are lacking. Notably, the spastic paraplegic gait is not present; the deep reflexes are not only not exaggerated but at times have even been difficult to elicit. I was unable just now to get the heel reflexes, as you saw, and at times it has been just as difficult to bring out the patellar reflexes. The Oppenheim, Schäfer, and Gordon signs also I have not been able to demonstrate today, while they were quite definite a few days ago, as Dr. Wechsler will agree. On the contrary, today she shows a doubtful Babinski on the same (right) side. Further, there is no sign of nystagmus, and the fundi are perfectly normal. Dr. Holden's report is entirely negative. All these cardinal symptoms of multiple sclerosis are lacking in this case, and yet the diagnosis cannot be doubted.

CORTICAL HEMI-ASTEREOGNOSIS WITH ACRO-AGNOSIS

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The two cases presented in this paper are instructive because of certain particulars in which they are strikingly similar; they are equally interesting, however, because of their dissimilarities. In one instance the symptoms were progressive in consequence of a malignant growth in the brain; in the other the symptom-complex was regressive, being the result of what was apparently a vascular lesion. Both patients, in addition to a pronounced cortical astereognosis, presented a loss of limb-knowledge, and it is to the existence of this acro-agnosis that attention is especially drawn.

Case I.[1] Patient: E. B.; age 50; female; married; white; houseworker.

This case is of interest not only because of its problem in diagnosis and localization, but because it brings up the question of the advisability of surgical interference.

HISTORY: *Present Illness.*—October 11, 1917, the patient came to the clinic complaining of loss of power in the left hand, general weakness, and headache. The condition was sudden in onset and progressive in character. Detailed questioning revealed the following:

On September 10, 1917, the patient noticed that the little and ring fingers of the left hand suddenly became weak, and she gradually lost power in them. A few days later the two fingers began to twitch. The twitchings lasted about one minute and came on several times a day. The weakness and loss of power extended to the rest of the fingers and to the hand. October 1, 1917, the whole hand twitched and there were cramp-like movements of the fingers. October 10, 1917, there was a convulsive twitching of the hand and forearm. The weakness slowly extended up the forearm and arm. Since the onset of the illness, the patient has suffered from frontal headache, mostly at night. She complained of general weakness and insomnia. There was complete loss of appetite, and a loss of weight of nine pounds. No vomiting. Patient urinates twice at night, and three or four times by day. She complains of precordial distress and general pains in both shoulders—more in the right. Vision seems normal, but she occasionally has dark spots before the eyes and flashes of light at night. The daughter says that the patient is irritable at times and occasionally depressed. Patient is right-handed.

Previous History.—Patient had measles in childhood and typhoid fever at the age of eight. The patient married at thirty years of age, had three children, all of whom are living; had no miscarriages. There is no specific history and none of trauma. No history of alcoholism or drug addiction. Menstruation began at the age of seventeen, was regular, of the four-week type, lasted four days, and was variable in amount. Menopause set in in 1913. She was perfectly well up to 1912, when she was operated upon for carcinoma of the rectum. An abdominal artificial anus was made, with perfect result.

In 1916, one year before the onset of present illness, the patient began to suffer from noises in the right ear, particularly on lying down; this lasted several months and then disappeared, apparently after treatment.

Family History.—Father is alive, aged 72; mother is alive, aged 70; one brother and two sisters are alive and well. One brother died in childhood. There is no history of cancer, tuberculosis, insanity, or epilepsy in the ascendants or collaterals.

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[1] Presented at the Clinical Conference, November 5, 1917

NEUROLOGICAL EXAMINATION

Voluntary Motor System. *Attitudes.*—Patient has left wrist drop; the left hand and forearm hang limply by her side.

Gait.—Shows slight weakness in the left leg, otherwise there are no abnormal phases.

Coördination.—There is no Romberg sign or other equilibratory disturbances. All non-equilibratory tests of legs and right hand are normal. Owing to weakness of the left hand none of the tests could be performed. The left hand showed adiadochokinesis and dysmetria.

Skilled Acts.—There is dyspraxia of left hand. There are no speech disturbances. Writing is normal.

Reflexes: (*0=absent, 1=slight, 2=active*)

(a) *Deep:*	*Right*	*Left*
Jaw-Jerk	1	1
Pectoral	1	1
Biceps	1	1
Triceps	1	1
Wrist	1	1
Radial	1	1
Ulnar	1	1
Patellar	1	1
Suprapatellar	1	1
Achilles	2	2
Dorsocuboidal	1	1
Periosteal	1	1
Crossed periosteal	0	0
(b) *Superficial*		
Ciliospinal	1	1
Supraumbilical	1	1
Suprapubic	1	1
Upper lateral abdominal	1	1
Lower lateral abdominal	1	1
Plantar and Its Modifications:		
Plantar flexion	1	0
Babinski	0	+
Chaddock	0	0
Oppenheim	0	+
Gordon	0	+
Schäfer	0	0

Abnormal Involuntary Movements.—No abnormal involuntary movements were observed.

Muscle Strength.—There is great loss of power in flexors and extensors of fingers of left hand, flexors and extensors of wrist and pronators and supinators of wrist.

Muscle Status.—No atrophies. There is hypo-irritability of the muscles of the left hand and forearm to both faradic and galvanic stimulation. There are no polar changes.

Abnormal Associated Movements.—There are no abnormal associated movements.

Nerve Status.—Condition is normal.

General Sensory. *Touch.*—Touch is normal, but tactile localization and discrimination were possibly impaired in the left hand.

Pain, Temperature, Vibratory, Pressure, and Muscle-tendon Sense.—These are all normal.

Astereognosis and Acro-agnosis.—Present in both extremities of the left side. With eyes shut the patient cannot tell the position of the limb and cannot hold it up as with eyes open. There is slight diminution in *posture sense* of left hand, forearm, and arm.

Cranial Nerves and Head. *Olfactory Sense and Nose.*—Normal.

Optic and Oculomotor Apparatus.—Pupils are round and regular in shape; 3 mm. in size; right somewhat larger than left; react normally to light and accommodation, both direct and consensually. Fundi are normal except possibly for a slight enlargement of the vessels. There is a slight lateral nystagmus. No ptosis, strabismus, or abnormality in conjugated movements at present.

Trigeminal and Mouth.—Normal.

Facial and Face.—Normal.

Acoustic and Ear.—Normal.

Glossopharyngeus, Vagus, etc.—Normal.

Spinal Accessory.—Normal.

Hypoglossus and Tongue.—Normal.

Mental Status.—The mental status is somewhat depressed. There is occasional irritability.

Cranial Morphology.—Normal.

SYSTEMIC. *Tegumentary.*—Abdominal artificial anus.

Glandular.—Normal.

Cardiovascular.—Blood pressure, 68 diastolic, 120 systolic; pulse 90.

Respiratory Apparatus.—Normal except for dyspnea on exertion.

Gastro-intestinal.—Abdominal colostomy.

Genito-urinary.—Normal.

Skeletal.—Slight scoliosis.

LABORATORY TESTS. *Wassermann* (blood). —Negative.

Urine.—Negative.

X-ray.—Negative.

SUMMARY AND DIAGNOSIS

The patient presents the following cardinal signs and symptoms: A history of gradually developing weakness and twitchings of left hand, headache, insomnia, and anorexia; paresis of left hand; left lower deep reflexes somewhat livelier than the right; left Babinski, Oppenheim, and Gordon; normal sensibility of left hand; astereognosis and acro-agnosis of the left hand. In view of the fact that there is a history of convulsive twitchings of a focal character and that the hand preserved intact all peripheral sensation except, possibly, posture sense, and that we found astereognosis and acro-agnosis, the lesion must be a cortical one. As the patient had a carcinoma of the rectum and at the present shows cachexia, anorexia, great weakness, etc., it may be assumed that there is a neoplasm of the carcinomatous type. The astereognosis shows involvement of the postcentral cortical area and the lesser motor involvement points to the hand area of the precentral convolution. The diagnosis, therefore is:

(*a*) Clinical: Neoplasm of the cortex.
(*b*) Anatomical: Ascending and superior parietal, impinging on ascending frontal.
(*c*) Etiological-pathological: Metastatic carcinoma.

Case II.[2] Patient: W. L. C.; age, 38; male; single; white; U. S. mining engineer.

HISTORY. *Present Illness.*—The patient's general health was excellent until March 10, 1917, when he awoke in the morning to find that he was blind in the right eye. The vision in the left eye was normal. On the day before the amaurosis appeared, he had ridden through the desert on a hard-gaited horse, being in the saddle nearly eight hours. He retired much fatigued, and in the morning found that he was blind. He could not see his hand held in front of his right eye, but could barely see light. At this time he had no other symptoms. He had no loss of motor power in any part of the body; no disturbances of hearing, of smell, or of general sensation, and was able to continue his business engagements with a slight inconvenience occasioned by the loss of vision in the right eye. He was at no time confined to bed. In the course of a week, some improvement was noticeable in the vision of the right eye, which progressively improved, until July, 1917, at which time he was able to read the headlines of a newspaper with either eye. On August 6, 1917, upon his return to New York, he consulted an oculist who informed him that he had suffered from a retrobulbar neuritis, probably due to an infection in the sphenoidal sinus. Consequently in September the sinus was opened and a gradual improvement of vision followed the operation.

In the first week of November, 1917—eight months after the onset of his visual disturbance—he began to notice lightninglike pain which ran down from the elbow to the fingers of the right hand. This pain was in all of the fingers, including the thumb; the palm was not particularly affected in this regard. The lightninglike pains would occur intermittently day and night. After they had persisted in the hand for about a week an intense surging pain made its appearance well back of the right ear and along the jaw to the chin, upon reaching which point it died out, only to recur at frequent intervals. These shocklike pains came on in the morning upon rising, or even, at times, awakened the patient; they would last about two or three hours, disappearing usually about ten o'clock in the morning. The frequency of their recurrence was about six per minute. The patient said they were often severe enough to make him wish to commit suicide. During the day they were absent, but returned at night-fall. He was occasionally awakened by them during the night. This pain lasted for a week or ten days. In the meantime the condition in the right hand had progressed to such an extent that the painful condition in it had changed, so that now the fingers and hand had a peculiar numbness. It was difficult for the patient to realize the position of his right hand, or what he might have in it. Thus, for example, he was not always sure upon putting his hand into his pocket that it was actually in the pocket, nor by the unaided sense of touch could he discern the nature of the objects which he might grasp. In other words, he developed a complete loss of power to recognize objects by palpation in the right hand. At no time have there been other symptoms of any kind, or other signs except those above described, which may be summarized as follows:

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[2] Presented at the Clinical Conference, January 14, 1918

1st: A more or less complete loss of vision in the right eye, which in the course of eight months returned almost to normal but which has shown some tendency to retrograde in the past few weeks.

2nd: Lightninglike pains in the fingers of the right hand, which in the course of several weeks were converted into a numbness, and finally into a more or less complete loss of power to recognize objects by palpation.

3rd: Intense shocklike pains along the lower jaw from behind the ear which began in the second week in November and lasted for about ten days.

Previous History.—The patient has lived most of the time, for the past thirteen years, in Mexico or South America. He has suffered from no frequent or severe headaches, no loss of consciousness, and no paralysis of any kind. About several years ago he began to have attacks of vertigo, especially when stooping downward, with a tendency to fall backwards, which, however, was always checked in time, and no fall resulted. He has had no discharge from either ear and no visual disturbance, and although he has always worn glasses, he can see without them. He has had a cough for several years, for which he has consulted several throat men. He has never had pneumonia or typhoid fever; but ten years ago suffered from malaria which lasted for three years, the attacks recurring about once a month and lasting for three days. In July, 1917, he had dhobie—a skin affection common in the tropics. He never sustained head or spinal injuries. He has been abstemious in the use of alcohol but has been a heavy smoker.

In February, 1917, he left New York for South America, and after an uneventful trip of three weeks arrived at Antafagosta, Chili, where he became engaged in examination of nitrate deposits. The patient has often been exposed to carbon monoxide in mines continuously for weeks at a time, during a period of one year; his last exposure to this gas occurred about two and one-half years ago, at an altitude of over 14,000 feet. It was a common experience for others who were with him during these exposures to be overcome, while he showed no apparent effects. His blood pressure has been peculiarly irregular. It has been found to be as high as 135 mm. and again as low as 103 mm. On December 13, 1917, it was 108 mm. systolic and 80 mm. diastolic. Upon one occasion he was poisoned by the bite of a scorpion, as a result of which he suffered from an edema of the pharyngeal mucosa and what appears to have been a temporary complete loss of vestibular equilibration; this disturbance lasted for several days but left no ill effects. He has also been bitten by the tarantula. Several years ago he was captured by South American insurrectionists; he successfully made his escape, but was forced to remain in a small open boat at sea with little or no food other than cocoanuts for seven days. He eventually reached shore in a condition approaching starvation.

Family History.—Father is dead; mother, living, and in good health; his grandparents, maternal and paternal, lived to about eighty years of age. In the immediate family there is one sister living and in good health. There is no epilepsy or insanity and no nervous or mental diseases in the family. The family history is also negative as to tuberculosis, cancer, and diabetes.

NEUROLOGICAL EXAMINATION

VOLUNTARY MOTOR SYSTEM. *Abnormal Attitudes.*—None.

Gait.—The gait is normal, although at times there is a slight limp on the right side.

Coördination.—Normal, both equilibratory, and non-equilibratory.

Skilled Acts.—All skilled acts of the right upper extremity are markedly impaired, although there is no paralysis in any of the muscles. This is due to the fact that the patient has not the proper limb sense, particularly in the fingers and hand of the right side. The left side is normal. All movements of the right upper extremity are distinctly improved if guided by vision, and distinctly impaired if without this guidance. There is a slight change of a similar type in the right leg.

Reflexes.—Measured by the reflex liminometer the threshold of the right knee-jerk is 8 oz., the left 12 oz. There is a slight increase of the deep reflexes of the right side as compared with the left. There were no pathological reflexes and no clonus was observed.

(0 = absent, 1 = slight, 2 = active)

(*a*) *Deep:*

	Right		*Left*		*Right*		*Left*
Jaw-jerk	1		1	Ulnar	1		1
Pectoral	1		1	Patellar	2	>	1
Biceps	2	>	1	Suprapatellar	2	>	1
Triceps	2	>	1	Achilles	1		1
Wrist	1		1	Dorsocuboidal	1		1
Radial	1		1	Periosteal	1		1

	Right	*Left*
Crossed periosteal	0	0

(*b*) *Superficial:*

	Right	*Left*
Ciliospinal	1	1
Supra-umbilical	1	1
Suprapubic	1	1
Upper lateral abdominal	1	1
Lower lateral abdominal	1	1

	Right	*Left*		*Right*	*Left*
Plantar	1	1	Oppenheim	0	0
Babinski	0	0	Gordon	0	0
Chaddock	0	0	Schäfer	0	0

Abnormal Involuntary Movements.—The fingers of the right hand are more or less in a constant state of motion or assume unusual attitudes. Mr. C. wears a mit on this hand because he says it makes it feel more comfortable. There are no convulsions, no twitchings, or other involuntary actions.

Muscle Strength.—There is no change in the muscle tone or in the strength of the right leg and arm, as compared with the left extremities. There is no loss of volume or contour in any part of the body.

Muscle Status.—The electrical reactions in both upper extremities are normal.

Abnormal Involuntary Movements.—None at present.

Nerve Status.—Shows nothing abnormal.

General Sensory.—*Pain, temperature, vibratory, pressure, and muscle tendon senses* are all normal.

Astereognosis.—There is a marked astereognosis in the right hand, also to a slight degree in the right foot. These impede movements in the hand and to some extent the movements of the right foot.

All elemental qualities of sensibility are retained. There is a slight retardation to pain and temperature in the fingers of the right hand and in the right hand itself. This condition is also present in a slight degree in the right foot. The faradic current requires 3 cm. coil distance in the right hand, and 2½ in the left to provoke a response. There is no subjective change in sensibility at the present time, except an abnormal sensation of numbness in the right hand.

Cranial Nerves and Head. *Olfactory and Nose.*—Normal.

Optic Apparatus.—Optic gives with glasses (correction) 12/20ths right, 12/20ths left, without glasses 6/20ths right, 11/30ths left. Optic disc is paler in the right than left. There are no hemorrhages or choking. There are no perimetric disturbances in vision at present.

Trigeminus and mouth, facial and face, acoustic and ear, glossopharyngeus, spinal accessory, hypoglossus and tongue sensibilities are all normal.

Mental Status.—Normal.

Cranial Morphology.—Normal.

General Systemic.—Shows no disturbance

of the tegumentary, glandular, cardiovascular, respiratory, gastro-intestinal, or genito-urinary systems.

RE-EXAMINATION. *January 12, 1918.*—He recognizes two cubes of three-quarters of an inch, and distinguishes between the heavy and light one (normal). He also recognizes a pair of bandage scissors, and distinguishes them from a pair of plain scissors in either hand. His recognition of weights and objects is more rapid in the left hand than in the right. He can recognize a small awl in the left hand in two seconds, which is not defined in the right for thirty seconds. He recognizes a rubber stopper in both hands accurately. Measuring tape recognized in the left hand in three seconds. A watch in the right hand not recognized in one minute. The watch and measuring tape were placed simultaneously in the two hands and were both recognized. He misinterprets material (flannel) in the right hand, and names it correctly in the left; silk is recognized correctly in both hands. In both hands calls a bone-handled knife bone, and a pearl-handled one metal. Calls a blotter a cardboard in right hand, in left a blotter.

Localization on back of right forearm shows a defect of 5 cm., and a defect of 2 cm. on back of hand. The ventral surface of the forearm shows an average defect of 2 cm.; ventral surface of the hand shows an average defect of 1½ cm.; tips of fingers show a defect of 2 cm., resulting in most instances in missing the finger altogether. At 45 cm. the patient is able to take hold of each one of the fingers on the right hand between the index finger and thumb of the left. At the same distance he makes a 4 cm. error in grasping the wrist, and a 2½ cm. error in grasping the elbow, and makes a similar error with the arm raised above the head 75 cm., and at this distance does not grasp the ends of the finger as well as at 40 cm.

Compression sensibility on the right forearm:

10 mm. Hg. ascending
20 mm. Hg. descending

This is normal.

Tested by the faradic current the distal phalanges palmar surfaces show:

	Right	*Left*
Little finger	3.0 cm.	2.75 cm.
Ring finger	2.5 cm.	2.5 cm.
Middle finger	2.5 cm.	2.75 cm.
Index finger	2.5 cm.	2.75 cm.
Thumb	3.0 cm.	3.0 cm.

Professor Holden's examination of the eyes, January 14, 1918, shows the following:

Corneas: Sensibility, normal.
Pupils: Size, right, 3.5 mm.; left, 3.5 mm.
Shape, right and left, irregular.
Light reflex, right and left, good.
Convergence reflex, right and left, good.
No diplopia, no nystagmus.
Mobility: Normal in both eyes.
Vision: With glasses, right, 20/30; left, 20/20.
Fields: Right, faint relative central scotoma; white test object seen everywhere but appears gray in the center of the field. Left field normal.
Fundi: Right discpale, chiefly in temporal half.

LABORATORY FINDINGS. *Wassermann.*—Alcoholic and cholestrin antigens negative.

Gonococcus Fixation.—Negative.

Ambard.—

D.	14, 28
Wt.	63, 24
C.	12.0
Vr.	.012
Index	486

Phthalein.—

1st hour . . .	64 cc.	27 per cent
2nd hour . . .	69 cc.	20 per cent
Total . . .	133 cc.	47 per cent

Lumbar Puncture.—Fluid came out clear, pressure not increased; 8 cc. removed. No reaction.

Spinal Fluid.—Cells: W.B.C. 10; R.B.C. 0.
Noguchi.—Negative.
Wassermann.—Negative in all dilutions.
Culture.—Negative.

Blood Count.—

Hgb.	114
R. B. C.	5,740,000
W. B. C.	8,400
Polys.	42 per cent.
Lymphos.	49
L. Monos.	4
Bas.	3
Eos.	1
Trans.	1

Stool: Twice negative for blood, ova, and parasites.

Blood Sugar: 95 mg.; Blood CO_2 — 29.75 mm. Hg.

Coagulation Time: 9 min., 45 sec.

Urine.—Normal.

X-ray.—Negative.

Differential Diagnosis.—There can be little doubt that here we are dealing with a pure astereognosis complicated by an acro-agnosis of the right side, more especially affecting the upper extremity. The absence of defects in any of the elemental qualities of somesthetic sensibility—as for example, in tactile, pain, temperature, muscle, joint, or vibratory sense—precludes the possibility of a lesion in the peripheral nerves, in the spinal cord, or thalamus. This limits the location of the pathological process to the cerebral cortex and, inasmuch as the superior gyrus of the parietal lobe is usually considered to be the seat of stereognostic sensibility, the lesion may be placed in the area upon the left side of the brain.

Far more difficult is the differential diagnosis in regard to the pathology. The clinical course of the case is of some assistance in this respect and distinguishes at once between processes which are progressive and those which are regressive in their tendency. Thus the relatively rapid and steady improvement would seem to rule out the possibility of cerebral neoplasm, atrophy, gliosis, or diffuse sclerosis. This brings up for consideration several pathological processes which may be, and often are, progressive in their course. Among these are: syphilis, encephalitis, meningitis, multiple sclerosis, toxic angiospasm, and the vascular accidents, hemorrhage, embolism, and thrombosis. Although the negative Wassermann does not necessarily exclude cerebral syphilis, the entire clinical picture does not favor this diagnosis, while its sudden onset and afebrile course are quite as much against encephalitis and meningitis. There is no marked multiplicity in the symptoms, so that, in spite of the pallor in the right eye, the absence of any part of Charcot's triad is a strong point against multiple sclerosis. Toxic angiospasm is always difficult to eliminate from the diagnosis of this condition; yet the absence of high arterial tension and the lack of evidence of angiospasm elsewhere in the body is distinctly against this possibility in the pathology of the disease. This leaves the vascular accidents to be discussed. The lack of evidence indicating high arterial tension or cardiovascular disease points away from hemorrhage and embolism. Thrombosis, the remaining possibility, is favored by the recognized low and fluctuating blood pressure and the coagulation time of the blood, which, by the method used, is relatively rapid. Whether his initial disturbance, *i.e.*, amaurosis in the right eye, may be interpreted on the same basis, is a question, and we must not lose sight of the fact that he suffered from an infection of his sphenoidal sinus. In my opinion, the condition may best be explained by thrombosis, since it is possible for the ascending parietal branch of the Sylvian artery to be thus affected and produce the symptoms I have described in this case.

DISCUSSION OF CASE II

Dr. Ward A. Holden.—The eye symptoms in this very completely studied case seem to me to be definite and hardly to be explained by thrombosis of retinal vessels. An eye, blind from thrombosis for two or three days, never recovers its vision. The history is that one morning the patient discovered that his vision was very poor in his right eye, although he could still recognize light. Some time later the condition was diagnosed as retrobulbar neuritis.

Acute affections of the accessory sinuses of the nose are frequent causes of the periostitis of the optic foramen which, by pressure, produces first a central scotoma and later almost complete blindness—a condition commonly called retrobulbar neuritis. This condition in this patient is indicated by the fact that his accessory sinuses were opened. It is not recorded that sinusitis was found, but after the operation vision gradually improved, as it usually does when there is no causative sinusitis and the etiology is obscure.

Infectious diseases like grippe are common causes of acute unilateral retrobulbar neuritis, while many of the bilateral cases are of toxic origin.

In this patient acuteness of vision in the affected eye increased, after some months, to 20/30, which it is today. The periphery of the visual field is normal, but there is a faint relative central scotoma and the optic disc is pale, particularly in its temporal segment.

Since this variety of optic nerve disturbance is fairly frequent and occurs usually without any symptoms on the part of the brain, it seems unlikely that in this case the eye symptoms were more than accidentally related to the cerebral condition causatively, although the onset of the two was nearly simultaneous; but even if the same causative factors were active in producing the two affections, it would seem to me that the ocular affection must be considered a primary retrobulbar disturbance of the optic nerve rather than an ascending disturbance due to blocking of retinal vessels.

Dr. Mark J. Schoenberg.—It is not clear to me what the vision of this patient was at the time he said he was blind. Did he have any perception of light at all?

Patient.—I woke up on the morning after a long, hard drive in the desert, still tired and found I could see nothing, not even my hand in front of my right eye. I had a dim perception of light but could not tell if it were day or night.

Thrombosis of the central retinal artery lasting for two weeks would suffice to produce a complete optic atrophy and total loss of vision. But the fact that the patient has regained so much vision proves that he did not have thrombosis of the central retinal artery. From the entire evolution of the case, it would seem that he had an exudate somewhere along the optic nerve, a retrobulbar neuritis. It would be lack of modesty on my part to discuss the process which took place in the cortex; however, I might say that if he really had an exudate in the optic nerve, the question is whether the same process did not also happen in that portion of the brain where Dr. Tilney localized the condition. If this is true, we may have to deal not with a thrombus but rather with several foci of exudates, and here the possibility of multiple sclerosis comes in.

One can have a retrobulbar neuritis of toxic origin; I have only recently seen one with intestinal intoxication and retrobulbar neuritis in a child of four years. If we exclude multiple sclerosis in Dr. Tilney's case, the condition may prove to be of toxic origin, and a careful examination of the bacterial flora and chemical reactions of the feces of this patient may throw some light on the subject.

Dr. Goodhart.—Assuming this case to be one of toxic origin, as thus it presents itself to my mind, the nearest analogy to the neurological symptoms is found in malarial infections.

The history of this case points definitely to previous severe malarial intoxication, whether alone, or as is more probable, provoked by a resistance reduced by physical exhaustion and, possibly added thereto, other toxins or organisms. The question presents itself whether or not the nerve symptoms manifest were not really of the nature of malarial invasion.

We do not know how toxins of the scorpion and tarantula injected into the human may affect the nervous system; we are cognizant of the fact, however, that both the toxins and plasmodia of paludism affecting the nerve centers can give the most variable symptomatology.

I have reported in full a case with amnesia as a symptom of invasion of severe paroxysms of malarial infection of the tertian type. The literature bearing upon the effect of malarial infection upon the nervous system demonstrates the variety of symptoms which may thus arise. Among those recorded are, motor aphasia, bulbar symptoms, hemiplegias, and a variety of abnormal psychic manifestations.

The pathological findings in some of the transient forms of malaria have shown in the cerebrum and cerebellum a hyperemia of the pial vessels, obstruction of the capillaries, their endothelium swollen with masses of changed red blood cells which pass slowly through the capillary vessels, owing to the increase in size of the latter, and their diminished elasticity, temporarily causing complete thrombosis. In some cases punctiform hemorrhages resulted. According to some observers, and this seems to me quite likely, and applying perhaps directly to the case under observation, neurological symptoms are due to capillary emboli or even emboli of the larger vessels formed by parasites; we also have the action of the toxins in other cases.

Furthermore, malarial toxins can cause disturbances during the interim in which no plasmodial symptoms are present. For example, vertigo, vomiting, ataxia, marked asthenia, hypotonia, and nystagmus, occurring fifteen days after the last paroxysm of malaria, in a case described by Forli, were regarded as due to toxins. We know that latent malarial influence has a tendency to become operative in conditions of exhaustion.

In my opinion the clinical manifestations in Dr. Tilney's case find elucidation in a condition analogous, identical perhaps, to what I have endeavored to describe.

Dr. Michael Osnato.—The fact that there is not homonymous hemi-anopsia in this case helps to localize the lesion, and there can be no doubt that it is in the cortex because capsular lesions with astereognosis usually give homonymous hemi-anopsia. The hemoglobin record and very prolonged coagulation time of the blood seem to point to a hemophilic condition. It is possible that changes in the blood of this patient have been brought about by his many infections, and possibly the scorpion bite is responsible for this. I know that rattlesnake bite can produce changes in the coagulation time of the blood and result in a hemophilia, in which these retrobulbar conditions, due to hemorrhagic pressure, may not be impossible.

The cortical astereognosis and acro-agnosis in this case appears to me to be more likely due to a slowly oozing hemorrhage in the posterior intermediate space, because of the character of the onset and the subsequent regressive character of the changes. The same applies to the amaurosis which came on over night and which could be explained by a retrobulbar neuritis due to the pressure of a hemorrhage since absorbed.

Dr. Louis S. Aronson.—It seems to me that a clinical picture, such as this one, might occur in uremia. In uremia and eclampsia one frequently finds a complicating retrobulbar neuritis of the optic nerve. Uremic amaurosis may pass off in a few hours or days and there need be no accompanying changes in the retinæ. There may be monoplegias and neuritides and no accompanying organic lesions in the brain to account for them at autopsy. The most astonishing thing is that the uranalysis may be practically negative and there may be no disturbance in the urea output in uremia.

This case is evidently one of some form of uremia, possibly due to the scorpion venom which acted on the brain through the blood stream, very much as does the poison of uremia, its action being sudden, short, severe, and toxic, and leaving nothing in its trail except a mild form of neuritis.

Dr. Frederick Tilney.—I desire to thank Dr. Holden and Dr. Schoenberg for calling attention to a differential point in this case which troubled me greatly in evaluating the association of the two lesions. It is possible that they were distinct and separate and not one clinical entity. The diagnosis of retrobulbar neuritis was made, and after the sphenoidal sinus was opened the patient's vision improved. The two pathological processes may not therefore be, so to speak, of the same piece. The blood picture and the blood pressure, together with the absence of any definite findings pointing to insufficiency in kidney function, would lead one to think of a thrombotic process rather than hemophilia. This, of course, is debatable ground. Where the blood pressure is low and variable I am accustomed to think of thrombosis, and so, with the general vascular condition of this patient and the fluctuations in his pressure, I believe we may picture a blood current which would predispose to thrombosis rather than a hemophilia, while the toxemia, suggested as the cause, seems too intangible and indefinite at the present time.

CONCLUSION

The general conception of astereognosis is often confused, although Hoffmann, who first introduced the term in 1885, and Landry, who first called attention to this condition in 1852, both had a clear and well-defined understanding of the nature of the disturbance. Landry, discussing the ideas which we acquire through the senses, particularly through touch, expressed himself thus: "That which we consider as a sensation in such a case is really only the result of the education which has taught us to correlate certain associations of sensations with certain ideas." In this sense, stereognosis represents a psychic elaboration and correlation of simple, elemental qualities of sensibility, both deep and superficial. Déjerine concurred in this opinion, stating that the stereognostic sense is a complex, a resultant, an association of various types of elemental sensibility, and no more a simple sensation than visual or auditory perception. On the other hand, in his clinical descriptions, he does not adhere closely to this conception, since he mentions the existence of astereognosis in tabes dorsalis, syringomyelia and other spinal diseases primarily affecting the elemental qualities of sensibility.

Roussy makes a particularly clear statement of the psychic processes involved in the recognition of objects by palpation. To recognize and comprehend an object, he maintains there are two different elements: (1) The conception of the form of the object, and (2) the comprehension of the object itself. The conception of the form of an object does not necessarily imply a comprehension of it, for it is possible to describe the object which is palpated without being able to give its name or define its usages. For disturbances in conception of form he employs the term, *astereognosis*, or, agreeing with Liepmann, Wilson, and others, *tactile agnosis*. On the other hand, defects in the comprehension of objects by means of palpation alone, he defines as *tactile asymbolia*.

Undoubtedly there is a distinction worthy of mention in these two special types of sensory conceptions. Both represent synthetic correlations of elemental somesthetic qualities, including tactile, pressure, muscle, joint, and perhaps temperature sensibility. Both require somesthetic memories, by comparison with which the form of the object is identified. Up to this point the psychic process may be called *stereognosis;* but when the object is recognized in connection with its name and usage, the process has gone one step further by bringing into action memories and associations from the visual, auditory, and kinesthetic spheres. Although the term *tactile asymbolia* serves to make the desired distinction, it is not an exact description any more than is *tactile agnosis*. Both of these terms, by giving the impression that the recognition of form depends upon tactile sensibility, fail to indicate the complex sensory synthesis which actually underlies stereognosis. *Tactile agnosis* should, therefore, be discarded as the synonym for astereognosis, while *astereosymbolia* would much more accurately describe those failures in somesthetic sensibility as a result of which the patient may recognize by palpation the form of a familiar object but be unable either to name it or comprehend its usages.

Stereognosis and stereosymbolia are pre-eminently correlational and synthetic activities of the higher cortical centers; it is unfortunate, therefore, that they have become associated in the clinical descriptions of peripheral lesions. In explanation of this, Roussy states that there is no astereognosis which does not involve a disturbance of some of the elemental sense qualities; in other words, that some degree of tactile, muscle, joint, or pressure sense disturbance is always present. This is not our experience and is particularly not the fact in the two cases above presented.

It is true that lesions in the peripheral nerves, spinal cord, and thalamus often give rise to defects in the ability to recognize the forms of objects; but in these cases there is always an attendant impairment in the elemental sense qualities. The explanation of such disturbances is found in defective conduction, not in the failure of cortical correlation. If, however, as a concession to clinical convenience, astereognosis is to be employed in the description of lesions in the nerves, in the spinal cord, or in the thalamus, it should be so qualified as to indicate that it is a peripheral astereognosis while pure or cortical astereognosis alone implies, in the strict sense, that disturbance in sensibility which results in the inability to recognize the forms of objects by palpation.

Cortical astereognosis is frequently attended by a loss of limb sense, *i.e.*, the patient is unable, without the use of vision, to realize the position of the arm or leg with reference to the rest of the body. That this is not a disturbance due to the impairment of sensory conduction may easily be demonstrated. Muscle, joint, tactile, and pressure sensibility may all be intact in the limb, yet, if blindfolded, the patient will grope with the hand for the affected part and be unable to find it. It seems reasonable to attribute this defect to failures in the cortical synthesis of limb posture, memories, muscle, joint, and pressure sensibility. This disturbance in sensation may properly be termed *cortical acro-agnosis*, to which we wish to draw especial attention as a frequent attendant of cortical astereognosis.

From the standpoint of localization, these two types of sensory disturbances may be regarded as indicative of lesions in the parietal lobe. Oppenheim, Monakow, Déjerine, Roussy, Bechterew, and Lewandowsky all agree in this conception of the localization of stereognosis. Spitzka still more specifically places this function of the cerebral cortex in the superior parietal gyrus. The common coincidence of acro-agnosis with cortical astereognosis makes it highly probable that the centers for limb-sense are in close proximity to those for stereognosis.

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EDITORIALS

SYSTEM IN RECORDING CLINICAL OBSERVATIONS

There are those who believe that they can get along without system. In medicine, system is regarded by some as a handicap to genius and excess baggage to originality. Such it may be to the illustrious few. Such it has never proved to be in the great coördinate activities which lead on to success. System is the basis of all organization, just as it is the secret of efficiency. All advances in knowledge are in direct proportion to the systematization of the facts cognate to each particular branch of learning. Every step toward such a systematization is a move in the right direction. Every obstacle raised against it or discouragement put in its path is to be deplored. Thus, for example, a recent editorial[1] in a medical journal inveighs somewhat bitterly against what it chooses to call the "Classifying Mania in Psychiatry." This apparently is directed against the highly commendable efforts of one of our most brilliant psychiatrists to introduce more general order and a more comprehensive system in the classification and diagnosis of psychiatric conditions. To designate these efforts as a mania is perhaps a loose use of language, perhaps dogmatic intolerance, or it may be even editorial exigency. The criticism, while it has its unfortunate aspects, is not, however, without value, since it affords the opportunity of emphatically commending this and all other efforts toward systematization. It also makes possible the rejoinder that the best way to encourage random and empirical treatment in any class of disorders is to discourage the attempts to introduce system in their diagnosis and classification. Furthermore, it may not be deemed good editorial advice which enjoins the psychiatrist to the quixotic pursuit of charging the windmill, admonishing him to abandon the introduction of system in any aspect of his subject, and to combat single-handed the entire domain of mental diseases with ergotherapy, psychotherapy, and endocrinous glands. No, it is the lack of system rather than overmuch interest in its behalf which hampers us. The evidence of this defect is all too obvious in the past, and the tendency to correct it none too apparent in the present. An instance of this is well shown in the attempt to study any disease as a composite clinical picture. For example, in a recent study of 150 cases of pineal disease recorded in the past hundred years, forty-six clinical details pertaining to this disorder might have been noted in each case. As a matter of fact, in sixty per cent. of the cases there was a record of only one-third of these details. Of the remainder, twenty per cent. gave notes on one-half of the details, and ten upon three-quarters of them. In no instance did any single case contain the record of all of these possible clinical details. This is not an isolated illustration, but may be found to hold true in the composite study of almost every syndrome. The reason for this defect is perfectly obvious.

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[1] *Med. Record,* xciii, No. 6, February 9, 1918, p. 244

There has not been, nor is there as yet, any common method of approach. Clinical observations are left entirely to the individual training and point of view of each clinician, and in consequence there is no common ground, nor uniform technique, to form the basis for a systematic method of case recording. It may be that this is an impossible ideal to attain. On the other hand, such an object may simply need the initial steps to start a movement for its achievement.

In the Neurological Department of Columbia University, such a form for the recording of clinical observations has been devised. This may not represent the best method, but it is one upon which the study and experience of three years has been expended and to which the deliberations of several other departments have been devoted. The beneficial results of its use are already evident, in that a group of men working in neurology is now able to use a common medium of expression in the discussion of their cases.

This movement toward system in clinical records has already been taken up by the New York Neurological Society. A committee, of which Dr. Charles A. Elsberg is the chairman, is endeavoring to formulate a uniform method for clinical records in neurology, which shall gain approval and be put into practical application in the several neurological institutions of New York City. Since the NEUROLOGICAL BULLETIN is primarily interested in the systematic recording of clinical cases, it is not amiss that the first editorial should be devoted to this movement. In fact the purport of these comments is an appeal for more system in clinical records in order to make them of greater value to ourselves as well as to those who come after us.

MORPHOLOGY AND CLINICAL NEUROLOGY

In no other branch of medicine has morphology played a more important rôle than in clinical neurology. In fact, the separation of neuro-anatomy and neurohistology from the clinical aspects of the subject cannot fail to be a disadvantage both in the teaching and research activities of neurology. In Columbia the entire subject matter pertaining to the nervous system is assigned to the Department of Neurology throughout the four years of the student's course. In this way neurological instruction is supervised and coördinated, with the chief object in view of making every course count in the practical training of the student. The staff of the Neurological Department, by this arrangement, has the opportunity to teach or carry on research not only in the clinical branches but also in neuropathology, neuroanatomy, and histology. Each member of the staff is expected, at some period of his service, to devote his attention to teaching in the first and second year subjects. This provides a general and broad training for the teaching staff.

Many problems in clinical neurology depend, in part, at least, for their solution upon the facts of embryology and comparative morphology. It will be the policy of the BULLETIN, therefore, to publish from time to time the results of such morphological studies. The first of these to appear is the work of Dr. Randal Hoyt, "Studies on the Morphology of the Gray Matter in the Brain Stem of Vertebrates by the Reconstruction Method." It is proposed that these short papers by Dr. Hoyt be subsequently collected as a more extensive and elaborate work. The first paper discusses the cuneate and gracile nuclei of the gorilla.

REVIEWS & ABSTRACTS

NEUROSYPHILIS: MODERN SYSTEMATIC DIAGNOSIS AND TREATMENT. Presented in 137 Case Histories. By E. E. Southard, M.D., Sc.D., and H. C. Solomon, M.D. 496 pages. 42 illustrations. W. M. Leonard, Boston, 1917. $5.00.

—*Lucidus ordo.* HORACE, ARS POET. 41.

Not long since I chanced to read the *Spectator* of September 5, 1712, the caption of which is quoted above. In this particularly lucid paper there are many valuable hints to writers of all kinds. Not a few of these hints might well have place in the armamentarium of the medical writer. Addison himself recognized in his own works two types of composition, namely, those which are written with regularity and method, and others which run out into wildness and go under the name of essays. "As for the first," he says, "I have the whole scheme of discourse in my mind before I set pen to paper. In the other kind of writing, it is sufficient that I have several thoughts on a subject without troubling myself to arrange them in order. Seneca and Montaigne are patterns for writing in this last kind, as Tully and Aristotle excel in the other."

There can be no doubt that "method gives light," and for this feature the new work on Neurosyphilis by Dr. Southard and Dr. Solomon is especially admirable. Dr. Southard's efforts toward systematization in the classification and presentation of psychiatric and neurological conditions are already well known.

This book, although primarily written for the general practitioner, cannot fail to be of use to the medical student, the neurologist, and the syphilographer. Its general excellence and value depend upon certain features in the method of its preparation. In the first place, the entire subject is presented by means of the case system. Concerning this method, Dr. James J. Putnam, who writes the introduction, speaks as follows:

"Undoubted as are the merits of the case system of instruction that has been so much in vogue in recent years, and excellent as is the modern supplementation of this method by the use of published records, the danger is still real that the student will have presented to him a picture of nature in disease that is too diagrammatic, too concise, with the result that while the task of memory is lightened through simplified formulation, the training of the doubting and inquiring instincts is often given too little stimulus and scope. In this book this danger is deliberately met through the casting of emphasis rather on the pluralistic aspects of the processes at stake than (primarily) on their unitary aspects."

Whatever the defects of the case system may be, it brings to the reader and student the facts of scientific observation in their most concrete and definitive form. The method has been used with great effectiveness in this book.

Another special feature is the systematization of the different forms of syphilis. This service in the present state of our knowledge is certainly a great one. The desirability of a simplified system is obvious and it is gratifying to find such a system in this work. The basis of the proposed classification is primarily the pathological alteration observed in the various forms of lues affecting the central nervous system. If the pathological process is confined largely or exclusively to the ectodermogenic tissues of the nervous system, this particular form becomes the parenchymatous or paretic neurosyphilis and has a well-defined syndrome of its own. On the other hand, the pathological process may affect the blood vessels and the meninges, in which case the form is known as the meningovascular type of neurosyphilis; again, both the parenchyma and the connective tissue elements in the brain may be involved with the result of producing the diffuse form.

The book is divided into six general parts, each discussing a special phase of neurosyphilis. The first part is devoted to the display of the several forms of syphilis affecting the nervous system, six of which are enumerated and defined. These forms are as follows:

1. Diffuse neurosyphilis. This form is meningo-vasculo-parenchymatous in type. Pathologically, it shows an involvement of the meninges, of the tracts in the spinal cord and also certain parts of the brain itself. In it, there may be evidences of an acute, inflammatory reaction as witnessed by a lymphocytosis in and about the spinal cord and brain or in one or more of the cranial nerves. Clinically, the symptoms are referable to disturbances in the sensory and motor systems as well as in the psychic organization.

2. Vascular neurosyphilis, which has the synonyms, syphilitic cerebral thrombosis and cerebral arteriosclerosis. In this form of the disease, there is an absence of meningeal and parenchymatous lesions. The spinal fluid in life may show a certain amount of lymphocytosis and possibly some plasmocytosis.

3. Paretic neurosyphilis with the synonyms, general paresis, dementia paralytica, and softening of the brain. In addition to the somatic and psychic changes of this disease, aortic sclerosis is almost constant. Another constant characteristic in paretic neurosyphilis is the plasmocytosis and lymphocytosis which is perivascular in its distribution about the small cortical blood vessels. There is also a certain degree of plasmocytosis and lymphocytosis which is meningeal in its distribution. Another histological characteristic is the granular ependymitis causing a 'sanding' of the ventricular floor. This latter is due to the fact that the limiting membranes of the nerve tissues are subject in these positions to multiple breaks.

4. Tabetic neurosyphilis, the synonym of which is tabes dorsalis.

5. Gummatous neurosyphilis, the synonym of which is gumma of the meninges or of the brain.

6. Juvenile paretic neurosyphilis, the synonym of which is juvenile paresis. In this condition there is cerebral atrophy, as in the acquired paresis. In the congenital form there are various stigmata of arrested development, including persistent thymus, hydromyelia, and in some cases a curious trefoil shape of the spinal cord. In juvenile paresis binucleate Purkinje cells emphasize the congenital origin of the lesion. In both forms of paresis plasmocytosis and lymphocytosis, pericellular in distribution and to a less degree meningeal, are found. Both forms also show a granular ependymitis.

Juvenile neurosyphilis may assume tabetic and diffuse characters, according as the lesions affect the dorsal roots of the spinal cord or involve all three of the constituents entering into the formation of the central axis.

The second part of the book discusses the systematic differential diagnosis and is valuable in its citation of disorders for which neurosyphilis may be and often is mistaken. This chapter should be especially useful to the practitioner as it serves to illuminate a number of pitfalls in diagnosis. Some of these conditions are displayed by illustrative cases as follows:

Paretic neurosyphilis frequently receives the diagnosis of neurasthenia. The differential diagnosis between these two conditions depends upon the testing of the blood for the Wassermann reaction which is unconditionally necessary. If the Wassermann reaction is negative, the diagnosis of paretic neurosyphilis is extremely improbable. It must be borne in mind, however, that a number of such cases have shown a negative Wassermann in the serum and receive proper diagnosis only after the spinal fluid has been examined. Certain clinical manifestations also aid in the differential diagnosis, for example, the pupillary reflexes and aphasic conditions. A clear insight into the condition and a continual search for recovery on the part of the patient point against a neurosyphilis and favor neurasthenia. Under this heading also is discussed the admissibility of a diagnosis of syphilitic neurasthenia and that of preparesis of Dana.

Paretic neurosyphilis may have many of the appearances of manic depressive psychosis. Even the circular course or recurrence of attacks in the psychosis is not decisive in favor of the manic depressive form. On the other hand, the laboratory tests of the blood and spinal fluid, together with such stigmata of neurosyphilis as may be found in the pupillary reflexes and the pupils themselves, changes in speech and the deep reflexes, suffice for the differential basis.

Paretic neurosyphilis resembling dementia precox: A case of this sort presented all of the schizophrenic proclivities, which in spite of this evident 'splitting' was obviously not dementia precox, since the blood and spinal fluid were both strongly positive to the Wassermann reaction.

Diffuse neurosyphilis may have all the appearances of paretic neurosyphilis during certain periods of the clinical and laboratory development. The difficulties of differential diagnosis under these conditions are marked, although in the main it may be said that the six tests for neurosyphilis all run slightly stronger in the paretic than in the diffuse form, while in the latter one or several of the tests may be entirely absent.

Tabetic neurosyphilis may simulate in many respects the taboparetic neurosyphilis, a combination of the symptoms of tabes dorsalis and those of general paresis. The importance of recognizing the difference between these two forms is considerable from the standpoint of prognosis, and even though the ultimate paretic picture is likely to retain many of the characteristics of tabes dorsalis, the appearance of the paretic phase indicates the severity of the involvement. Tabetic neurosyphilis, on the other hand, may be so atypical clinically as to show no single symptom justifying the older clinical diagnosis of locomotor ataxia. Such, for example, are the monosymptomatic and oligosymptomatic cases of tabes dorsalis, and those in particular which chiefly if not entirely localize the lesion to the cervical region of the cord, giving rise to what is generally known as cervical tabes.

Neurosyphilis in any of its forms is not to be ruled out by a negative serum Wassermann reaction, for in a certain number of cases, variously estimated from three to five per cent., the spinal fluid Wassermann may be positive. On the other hand, a positive Wassermann reaction of the serum may be present while a negative spinal fluid reaction is found. This frequently occurs in neurosyphilis of the vascular type. The other signs in the fluid are frequently positive, that is to say, the pleocytosis, the increased albumen and globulin. Diffuse neurosyphilis and paretic neurosyphilis are not infrequently ushered in or attended during some period of their course by epileptiform seizures. These attacks may be generalized or Jacksonian in type. Such likewise is the case with regard to motor aphasia in these two forms of neurosyphilis. . The aphasic involvement may be transitory and accompanied by transient or fleeting paralyses of various parts of the body. Under these conditions, the laboratory tests will give the proper indications concerning the character of the disease, and the tendency of these tests to run strong should be considered in favor of the paretic neurosyphilitic type.

Neurosyphilis may present itself in the form of a spastic paraplegia; that is to say, the syphilitic spastic paraplegia of Erb.

Neurosyphilis with the appearances of muscular atrophy: Under these conditions other signs and symptoms are present which remove the disorder from the class of the dystrophies. Sensory disturbances and speech defect, together with pupillary abnormalities, combine to make this distinction clear. There is a question as to the possible relation of syphilitic muscular atrophy to amyotrophic lateral sclerosis. Some years ago, Spiller claimed that such a relation existed and the authors think there is some justification for this view.

Congenital neurosyphilis is apparently capable of causing feeble-mindedness without any special tendency to progression or any tendency towards vascular insult.

Secondary syphilis, in about a third of all cases, is marked by the signs of neurosyphilis not unlike those of a fully developed paretic or diffuse neurosyphilis. These signs are sometimes those of the clinical order but on the other hand may be confined exclusively to the laboratory tests, and the case itself may present no clinical symptoms. In this connection the question naturally arises how early clinical evidence of neurosyphilis may set in after the initial lesion. According to Craig, one case of brain syphilis was found occurring one month after the infection. Frye cites a case of tabes dorsalis developing in six weeks, while Craig states that he has had three cases of brain syphilis occurring within six months and six within a year of the infection.

Certain puzzles and errors in the diagnosis of neurosyphilis are considered in the third part of the book. Here are considered such difficult complications as hysterical fugues, cerebral gliosis, exophthalmic goiter, encephalitis, pseudoparesis of alcoholic origin and also due to diabetes, acute and chronic alcoholism which may obscure the diagnosis, brain tumor, multiple sclerosis, and senile arteriosclerotic psychosis. Even such unusual conditions as Huntington's chorea and pernicious anaemia may make their appearance to confuse and complicate the situation.

In the fourth part, a particularly interesting phase of neurosyphilis is discussed, namely, the medicolegal aspect. This furnishes a valuable contribution. In this chapter are shown the dangers and embarrassments arising from unsuspected neurosyphilis in influential public persons, as well as the dangers due to changes in conduct and character of those occupying positions of trust and responsibility. Marked character changes should always bring such individuals under suspicion and at the same time necessitate further investigation into a possible luetic basis of their disorder. The question of the legal responsibility of neurosyphilitics, early delinquents and defectives, *paresis sine paresi* in the habitual criminal, together with the important consideration of traumatic exacerbation of neurosyphilis, the duties and attitude of the physician to the family of the neurosyphilitic, and the advisability of marriage are discussed in the light of illustrative cases.

The fifth part deals with the treatment of neurosyphilis in all its various forms and shows the results in twenty-four cases illustrating all varieties. Concerning this phase of the subject, the authors speak as follows:

"We are confident that no one can now successfully make a differential diagnosis between the paretic and the diffuse non-paretic forms of neurosyphilis in many phases of either disease, even with all laboratory refinements. If this be so, it is improper not to give the full benefits of modern treatment to all cases in which the diagnosis remains doubtful between the paretic and the diffuse non-paretic forms of neurosyphilis. We ourselves advocate modern treatment, not only in the diffuse, but also in the early paretic forms of neurosyphilis."

"Neurosyphilis and the War" is the topic of the sixth part of the book. Here are presented fourteen cases selected from the British, French, and German contributors to war literature, all of which seem to draw attention to the unusual interest of the neurosyphilitic problem in regard to the war. One case is that of a young lieutenant who had tabes dorsalis and was *shell-shocked* into paresis. This proposition, however, is properly questioned by the authors. The hypothesis which they advance is that the explosion in some way indirectly caused an alteration of the living conditions of the spirochetes and thus permitted the development of paresis. Latent syphilis, converted by shell-shock into tabes dorsalis, neurosyphilis activated by trench life or produced by 'gassing', latent epilepsy made acutely active by syphilis acquired during the war, and syphilitic root sciatica developed as the result of work in the war zone, are some of the various neurosyphilitic conditions which the authors believe to be precipitated or aggravated by the stress and exigency of war times.

The lessons of this book are summed up in a general way as an effort to give special emphasis to three points, namely: "1. The unity-in-variety of the phenomena of neurosyphilis. 2. The value of a hopeful approach to the therapy of all cases of neurosyphilis, even the paretic form. 3. The value of applying syphilis tests to every case of neurosis or psychosis."

An appendix gives in simple form the special laboratory tests for neurosyphilis, while a second appendix provides a summary of the common methods of treatment used in cases of neurosyphilis. Appendices A and B give with admirable directness the necessary instructions for laboratory technique and treatment needed by the student.

In the midst of so much excellence there should be something less obviously good, even for variety's sake. One may indeed run the risk of being captious, for most of these matters are of minor importance, yet it is to be regretted that a book so good intellectually is so poor in its manufacture. American publishers still have something to learn concerning the respect and treatment due high class medical works. The illustrations are good but not numerous enough. The absence of references to the literature cited is always a serious defect in any book. References in the text acquire added value and serve a broader purpose when the place of their original appearance is indicated. It is to be hoped that the authors will take this matter in account in future publications. The lack of uniformity in the recording of clinical facts and observations constitutes one of those almost universal and lamentable defects in medical science to which more extended reference is made in another place. (See page 86.)

And then—why stop with Baalim and Ashtoroth? Why not the entire list of big and little devils? Surely none too many! Why, indeed, introduce the Great Architect of Hell at all if he be not permitted to vent the full volume of his malignant blast? And again—is there any particular significance in attaching a quotation from *Paradise Regained* to the chapter on the treatment of neurosyphilis? It must be so, for all of the other chapters, with one exception, have quotations from the memoirs of that other reputed refuge in *Paradise Lost.* Perhaps, in general it may be well to accept Dr. Southard's apology to John Milton for his borrowings from the two *Paradises.* Otherwise, it might be necessary to reveal some 'mechanism' in the explanation of this somewhat unusual congress. To one of simple mind and of little leaning toward the psycho-analytic complexities, the 'mechanism' of *irrepressible erudition* might suffice; such, for example, as was displayed in my own case when I spent several hours searching through the *Spectator* to find a motto, erudite and, for that reason, worthy of introducing the discussion of this good book. The authors are to be congratulated and none can read their work without gaining much enlightenment in the subject of neurosyphilis.

F. T.

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BORDERLINE PSYCHOSES

STUDIES IN MENTAL HYGIENE

By F. M. HALLOCK, M.D.
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NEW YORK CITY

In a large clinic such as that of the neurological department of Columbia University we see daily a type of case familiar to every neurological clinician and recognized by him in a general way under the name of "Borderline Case." These cases constitute such an immense problem in neurology that they deserve the broadest possible study and for this reason a series of them will be presented in this and succeeding numbers of the NEUROLOGICAL BULLETIN. It is our purpose to show that, even with the facilities we now have, a great deal may be done for these patients, and a great deal more ought to be done for them. Among these patients are the cases of mental disturbance which, although they cannot be called insane, yet are not normal. They do not always fall into any fixed groups. They may be diagnosed as simple depression, incipient dementia precox, mild manic depressive insanity, or manic depressive make-up, undifferentiated depression, and so forth. It is often very difficult to classify these patients and sometimes difficult to make a diagnosis. I recall the case of one patient in which manic depressive insanity, epilepsy (with psychic equivalent) and hysteria were equally supported. Another case in which it took time (several weeks) to make a decision between epilepsy and hysteria; another in which psychopathic personality, epilepsy (psychic equivalent), hysteria and dual personality were about equally supported.

Aside from this class of cases are others, clinically somewhat allied to them, also social burdens and misfits, the so-called psychoneuroses which include the hysterias, anxiety neuroses (a very large group), the phobias, obsessions, and compulsion neuroses.

These patients are proportionately very numerous and are a great social and economic factor; they are a burden to themselves, to their families, and to society in general; sometimes they even become a menace. I am sorry to say they are too frequently regarded in the clinics and elsewhere also, as a mere nuisance. A great many of these cases may be saved which now merely drift about or frequently go from bad to worse. It is the aim of these studies to present types of these groups and to demonstrate the possibility of reaching these patients.

The better I become acquainted with the life histories of such patients, the more I am impressed with the moral and social difficulties with which they are beset, especially in their childhood, and the more surely I become convinced that it is a question of reaching the individual early enough to be of help. These studies might aim to encourage us in the direction of improving our methods and means of handling such cases. I am more and more impressed with our ignorance of the real factors; they are all struggles for an adjustment to life and there is plenty of evidence to bring this into view as a clinical fact.

I shall present first two cases diagnosed as early or incipient dementia precox, very much alike in type. We see a great many of this type of patient. I cannot regard either of these cases as hopeless even though they have progressed as far as they have—less hopeless, even, if we could have begun to work with them earlier. Following these, Case 3 presents a severe compulsion neurosis. All of these cases are chosen from Vanderbilt Clinic material. In succeeding papers we shall select for publication some of the more familiar types such as hysteria and the psychoneuroses.

A BORDERLINE CASE SHOWING SYMPTOMS OF DEMENTIA PRECOX IN ITS INCIPIENCY

Case I; No. 47637; A. B.; age 21 years; male; a clerk; single.

The patient came to the Vanderbilt Clinic giving the following symptoms:

History.—*General Complaint.* Sensation of creeping over the top of the head which "keeps me from doing my work. Pain in touching my head. I feel very funny, as if I'd lose my mind."

These symptoms have existed for eight months with a history of gradual onset. There is no history or evidence of syphilis, no history of trauma, alcohol, tobacco, or any diseases. The physical examination is entirely negative.

Present Illness.—The patient speaks with great anxiety about "funny feelings in his head," "creeping in the scalp," "queer feeling I cannot describe," "I have no interest in anything," "I cannot do my work." "I have no ambition." These feelings came on gradually over a period of six or eight months. "If a picture (*x*-ray) could be taken of my head the cause might be found." "About three weeks ago, every time I took a step I could hear something cracking in my head. Sometimes I have a feeling as if my head was separating, a funny feeling, I can't explain how I feel."

When asked if he hears voices, at first he says he hears his name called, and so forth—but later says that these are his own thoughts and that he does not actually hear voices.

There are no hallucinations of sight, orientation for time, place and person is normal, and there are some ideas of reference. The patient speaks of having had ideas of the family annoying him at home, even of his brother trying to harm him, and once, when he had indigestion, he imagined his sister "doped" his food, but he says, "I *imagined* it." "I knew it was my imagination and have to laugh now for such thoughts that used to go through my mind."

"I can't stick to a job; it bothers me terribly that I can't." (Question: "Why?") "I get discouraged quickly; I make mistakes and it is due to that feeling in my head." "Sometimes I will be spoken to and can't seem to understand, I'm not alert, you've got to be alert to work in an office." The patient volunteers sexual conflicts, says he never had intercourse, and speaks of spontaneous ejaculations.

Previous History.—He never recalls being ill, says he was a healthy child, and "very ambitious." He began school at six years of age and continued until the age of fourteen. He worked to help his father between school hours. He had good marks at school and enjoyed his school life. At fourteen he went to work for his father where he remained three or four years.

"My father was establishing me in the candies and fruit business. The business succeeded and after a time I made thirty or forty dollars a week. We were dispossessed, and did not think it worth while to set up elsewhere. After that I could not get settled in any business. Got a job in a factory where I remained two months earning eight dollars a week. I got sick being indoors; got a job as a driver and earned eighteen dollars. I remained in this place one year and left on account of hernia for which I had an operation. Then I learned stenography, and hung on to various jobs, perhaps half a dozen, earning ten or twelve dollars a week. Then I went into an auto concern as a typist and stenographer earning sixteen dollars, where I remained one month. I left this place because I didn't know about the automobile business. The last job I got one week ago at a salary of sixty-five dollars a month. I feel so queer—my head—it's very hard for me to stick to my work."

The patient gives a straightforward account of himself, is manifestly worried over his condition, and does not try to evade the examiner. He is puzzled for words to describe his peculiar feeling. Insight and coöperation are good.

Dr. Sylvester Leahy who also saw this patient has made the following notes:

"The patient still continues to believe, at times, that his relatives are against him; he doesn't speak to his sister because he thinks she is against him. If he sees people talking together, even strangers, he believes they are talking about him. Since the onset two years ago, he has expressed all sorts of physical complaints and has changed positions very frequently. He has always been of a quiet, seclusive make-up, does not care to go out much, and never did. In the diagnosis dementia precox should be considered as a possibility."

At my second session with the patient, a week following the first, he continues as follows:

In response to a question "What do you think is the trouble with you?" he says, "I think the cause of my trouble might be that I ought to have sexual intercourse." The patient tells me with some emotion that he masturbated between the ages of eleven and fourteen; he thinks that this might have been the cause of all his trouble. Later he tells me he went to a prostitute a year and a half ago and again with considerable emotion states that he "could not do anything." He was unable to perform the sexual act. He volunteers, "she is a miserable prostitute, a paid woman, and I had a kind of disgust for her." Again, later, he tells of another visit to a prostitute three years ago when he was also impotent. This time he was afraid because the thought of disease came to him and he could not feel sexual desire. Yet, he says, "I feel much sexual excitement for other girls that I see."

At the next session (two or three days later) he continues (practically all this production is spontaneous).

"I think a lot of my trouble is due to the environment at home. I have to stand an awful lot of abuse; sometimes I feel like leaving home."

(Question, "Who treats you badly?") "My mother shows favoritism to my brother and sister and I do not speak to them. I have a constant fear of going insane and am always miserable about myself; I am afraid that I can't hold the job I am now in although it is not at all a bad job. My head does not allow me to be pleasant even; my father treats me well, fine, but my mother shows favoritism to my brother and sister."

(Question, "Are you fond of your mother?") "Yes, very. When I'm talking with people I get such a queer feeling in my head, I feel as if I shall lose myself and have to stop talking. I feel queer, and I feel they think I'm queer, and then I lose all confidence. The moment I take dictation, in the beginning, I have no confidence that I can do the work but when I get started I can work all right."

The patient continually reverts to his home conditions as being intolerable. "If I could *talk* at home; if they would let me talk to them, I'd feel better. I say to myself, am I not a good person that they do not talk to me? Did I ever commit a crime that they should ignore me? I like my mother! My sister orders her around as if she were a servant! That hurts my feelings! Two years ago I accused my married sister of poisoning the food. When I see her I regret it so much but she is really good to me! Maybe it's all because I don't have sexual intercourse, that I feel so bad; it's a natural thing. I reason this out to myself time after time. I am self-conscious."

At the next session the patient brought the following typewritten account of himself: "The evening I left your office I went home, my mother was very anxious to know what the result of the interview was, and what you thought about my condition. I told her that I told you about the conditions at home and that you thought it would not be a very wise thing for me to leave home. My mother assured me that when I feel that I am not welcome at home and that everybody has hatred toward me that it is only my imagination. This statement made me feel very good; I went to bed and slept sound. That night I dreamed that I had met a moving picture actress (I had not asked the patient for dreams, nor said anything to him about them) whose name I could recall without the slightest hesitation. I walked along the street with her and when I passed my house and looked through the window from the outside I saw my whole family and they looked so down-hearted to me that I refused to go in with her, and she took me to her friends and introduced me to a number of them. The dream was so clear to me the next morning that I remembered her name and everything that happened. When I got out of bed that morning I felt very good. As soon as I got to work that morning and worked about one hour I got that funny feeling in my head. I felt very queer all morning, and in fact I felt very badly all day. I went home and after supper I took in a show. While sitting there the moving picture machine stopped at the end of the Fourth Act and we had to wait until the operator got his film into shape again. While the intermission lasted I got into conversation with a young lady which I enjoyed very much; we spoke throughout the show, and then I walked her partly home. The next morning before getting up something made me feel that I didn't want to go to work. I finally got up, ate my breakfast and went to get my train. While traveling I felt very good, whistled different songs, and felt good in general. Before I entered the office right after lunch I felt something drawing me back and when I entered there were men other than the office force in the office; these men are some of the executives from our main office in another city and this made me lose control of myself and I felt that if any of them should have spoken to me I would not be able to answer them intelligently. To make myself not feel that way I say to myself 'Well, these men, they are much older than I am and naturally they know more than I do'. Before I got up this morning, while laying in bed I said to myself 'Why should I suffer so much?' (Then an obliterated sentence—'Oh, if I could only end it all.') If this is how I am to go through life, then life is not worth living. When there is conversation in the office I find it very hard to do my typewriting, hence I make a number of errors; in fact, this trouble which makes me think about myself causes me to make a number of errors. If my trouble could be remedied I am certain that I would be able to do my work with much more care, and work would be to me a pleasure instead of what it is."

To the paragraph which follows the patient called my special attention as being in his mind the real cause of the whole difficulty. "I would say that my trouble and inability was caused from worry and too much thinking about one subject all the time, and about my troubles of the past. Also, one and a half years ago in the summer while at work in the early part of the morning I became dizzy, everything before me seemed to move back and forward. My employer suggested that I take a physic, which I did. I came back to the office and became temporarily blinded. The shipping clerk took me home and when I went on the car I felt myself weakening. I I heard him say faintly 'Do you want to get out?' Then I felt myself weakening and collapsed. In the afternoon I found myself in the hospital; they diagnosed my case as being 'Anæmia of the brain—epilepsy'. This morning when I got up I felt the same way as I do ordinarily; no ambition, etc. While in the office this morning I looked up the definition of the word 'epilepsy' and I found it to be 'crazy'. Well, part of the morning I was thinking whether my condition would drive me insane. When I have a hearty laugh, which is very seldom, my head feels very raw, and I feel like worms creeping around in it. At times I begin to think that my brain is rotting."

SUMMARY

Here we have a boy who complains principally of some peculiar feelings in his head; has some questionable hallucinations of hearing and some more definite ideas of reference. For a period which he gives as eight months (but which is probably longer) he has been suffering rather acutely and with increasing severity in his symptoms. There is a history of inability to hold a job, he cannot keep at his work and has gone from one job to another over a period of several years. He is not adjusted to his home conditions, and has even had something like a delusion in connection with his older married sister. Certain of his symptoms are of a characteristic precox type, such as the feeling that his brains are rotting, and the whole history taken in general is, of course, that of an early precox. But the boy's account of his social difficulties is spontaneous and plain; it is a sexual affair. He does not understand it but he has located it in a general way. His insight is fairly clear, and his coöperation so far as I am able to judge of it at present, is excellent.

This completes the history of the case so far as I have gone with it. The nature of the boy's conflict is certainly plain. He has shown in his subsequent visits an attitude of ready coöperation, he has a great deal of insight, and my impression is that with favorable conditions under which to work with him, this patient will clear up entirely. Whatever label we put upon his disorder, the case is one of social maladjustment. The present facilities for solving problems of this kind are still unfortunately limited, and it is with the purpose of calling attention to the necessity of developing the possibilities in dealing with such conditions that this case has been cited.

A SECOND CASE OF SOCIAL MALADJUSTMENT ON THE BORDERLINE OF DEMENTIA PRECOX

Case II; No. 39027; R. Y.; age 22 years; male; born in Russia; operator; single.

History.—*Present Illness.* The patient complains of nervousness, he does not sleep, and has a poor appetite. He dates his illness from the time of an operation four years ago. He says he "is nervous, can't talk out what he means." "I feel like falling." "I can't go among people." "I hang my head." "I get only two or three hours sleep; sometimes I hear my mother call me; I will sit silent unable to reply to her." (The patient is not here referring to hallucinations.) "Sometimes I hear voices telling me to do one thing or another, and if I wouldn't do it, something would happen to me."

The patient thinks that he never should have been operated upon, that there was nothing wrong with him, that the surgeon who operated was "no doctor." He went to see him and told him so. The patient has changed employment only twice in four years. "In one place the boss hired a lot of girls and wanted us to work on equal pay, but I left. I have not worked for three weeks because one week was cold (this statement is probably correct) and the second week the foreman told me to wait and he would later employ me at my old wages of seventeen dollars a week." At the beginning of the third week the patient talked to the foreman about his old wages. This matter was arranged satisfactorily, and he had commenced work when the "boss" came in; they quarreled about his having been late on the occasion of his first employment. The patient insisted on piece-work, finally said it was too cold to work, anyhow, and left his job.

Voices compel him to remain unhappy; they call him names if he enjoys himself; he tries to break their spell, and says, "Everytime I have a fight between me and I don't know who else, they fool me. When I try to do the best, and be the best, they are dissatisfied, and say 'Who the hell is he?' " The voices make him angry. The patient also has visual hallucinations. He sees himself no longer as R. Y. but as the Devil. "I see him and he is myself, and if I don't look at him I feel better. He is my own shadow, and it is as if he were in a looking-glass; the Devil shows me when I do wrong things."

All this is given in English, but I get the impression that he would express the same ideas quite differently in his own language. The patient has a great deal of insight and is coöperative. I am impressed with this fact. Upon superficial examination the patient showed me catatonic signs. He spoke English very poorly and owing to the difficulties of language between us, I referred him to Dr. Adolph Stern. The following is transcribed from his notes on the case.

"The patient's early recollections are that he went to a rabbinical school between seven and eleven years of age. He did not care to study, was very troublesome and obstinate, though as a rule he was retiring, shy, and given to day-dreams. At eleven he went to work in a factory. The patient can remember, even at this early date, feelings of hostility for his father because the latter, he believed, did not support the family well and because the patient had heard that his father had been unfaithful to his mother. Also there was present a feeling of jealousy toward his oldest brother, the mother's favorite.

"On his arrival in this country, he was exceedingly shy, retiring and introspective. At thirteen years of age the patient began to masturbate though he gave a history of an active sexual life between the years of nine and eleven. The masturbation was accompanied by many conflicts both moral and hygienic, for a boy, whom the patient loved and respected, had told him that masturbation brings on the worst kind of sickness and "that once you are in it, you can't get out of it." This boy also told the patient that a masturbator could be identified by a peculiar facial expression. All this decided the patient to stop masturbating. He did so and there developed cardiac palpitation, anxiety states with dizziness and insomnia. This lasted on and off for six months and the patient felt perfectly well, 'even better than before'. During the period of illness he worked very irregularly losing one job after another, but when he felt well became steady in his work.

"From his fifteenth year, the tendency toward isolation and introspection, always present, became even more marked. His associates became fewer in number, nor could he feel himself on very intimate terms with members of his family. He ascribed this estrangement from his boy associates to two things; first, he was now grown up yet was not at all Americanized and felt out of place with the boys; second, the dread that the boys would find out that he was a masturbator. For, though he had resolved to stop, and had succeeded to some extent, yet the temptation to perform the act was at times too strong and the patient yielded.

"He went along, up to his nineteenth year, managing fairly well, in spite of occasional attacks of dizziness, fainting spells, and cardiac palpitation. Only for very short periods, however, was the patient psychically (emotionally) at ease, that is to say, free from conflicts.

"In 1913 he was operated on for appendicitis. Although he made a rapid recovery yet it was three months before he was able to resume work owing to weakness and fatigue. He then started to work, but at the end of nine months had to stop, because the former symptoms of dizziness, cardiac palpitation, and fainting spells again returned. At this time an additional, distressing symptom was a sensation as if the ceiling and walls of the room were closing in on the patient.

"In the fall of 1915 his oldest brother died. Following this death, the patient's condition became much worse. He went to see several physicians, but without relief. Early in 1916 the patient went to a Scientist who, the patient states, hypnotized him, though he resisted somewhat.

"The symptoms which prompted the report of this case and which make it of special interest from a diagnostic, and possibly from a therapeutic standpoint, began to manifest themselves a short time before he sought treatment at the hands of a Christian Scientist. The patient at times felt a sense of unreality of things. Although he knew no change had taken place, yet he felt that things were different, not real. Things seemed changed. While at a doctor's office, for instance, it appeared to the patient that it was not a doctor's office, but some strange, unfamiliar, peculiar place, although he was fully aware of the actuality of the situation. Soon thereafter the patient began to hear voices, which at times were stern and harsh, bidding him to do one thing, while another voice told him not to do that. Often, while at work, and feeling very nervous and 'bad', he tried to suppress these feelings, and he felt that he could look into himself, into his own heart, where these thoughts came from, and tried to crush out these bad thoughts, at whatever cost. (The patient realized that these impulses were all parts of himself, and I explained to him that these were his own conflicts against masturbation. The patient accepted this with manifestations of conviction.) The patient fears that this arguing with himself might make itself manifest in his face and be noticed by people who would think him crazy. He also 'felt' a voice, telling him to keep away from people because he was 'bad'.

"That which strongly savors of a dementia precox is evidenced by the following; it pertains to his attitude in reference to the Christian Scientist, who, the patient states, influences him very strongly. 'The Scientist says I have no body, that I am a spirit, and that he will be behind me and make me do things. That he will correct everything that is wrong in me; he can make my bowels act, when they refuse to work for me. Sometimes I believe all this; at times I don't'.

"The patient is conscious of the fact that he laughs at times foolishly. He states that he has done this for years and that for the past year and a half it has become even more marked. He explained it in this way; that he felt that now he was very sick, that this world was no longer for him, and that nobody can help him now and he decided to laugh at it all, like one who drinks and forgets all his troubles."

"While there is a doubt as to the diagnosis in this case, yet, even though we call it a dementia precox, we cannot feel that it is a dementia of the institutional type for the patient gives his active coöperation, and is fully approachable. Moreover, there is a realization on his part that these voices, at least some of them, are parts of himself, and to us it is evident that some of them are reproductions of his early conflicts. This is well illustrated by the statement of the patient that at times he tries to hold up his head and walk erect, at the same time telling himself that he is not *all* bad, and that there is still some good in him, when another voice says, 'You are all 'bad'.

"This patient presents several interesting features. He has been sick now about four years, and for the past three and one-half years has shown definite hallucinations, the true nature of which he only partially knows; yet he seeks relief from the tormenting conflicts which they cause him. In this respect he differs from the average dementia precox patient. Another well-marked difference lies in the patient's active coöperation with the physician in his desire to get well. He manifested at the time of the examination, a perfect frankness regarding his symptoms. He told of his delusional state—that a Christian Scientist who had been treating him was now influencing his every thought and act, making him see that which his own eyes assured him did not exist; he stated he knew this did not actually exist and should not exist, and sought relief from this 'influence'. At times, during the narration of the struggle against the 'influence' of the Christian Scientist, the patient spoke of this as if describing an actually existing state of affairs, displaying during the narration an intense feeling of anger at the scientist for the trouble he was causing the patient. After the narration was completed, his face clouded over with mingled anger and despair, and with beads of perspiration on his face, he looked at the examiner and said, 'It's all so strange, I wonder what it means. Sometimes it seems so real to me, but when I stop to think, I feel it can't be as I am telling it to you'.

"Another point of interest is the comparatively normal life the patient has been able to lead, in spite of the intensity of the symptoms; as manifested by the ability of the patient to continue at work, though less regularly than before the onset of his illness. His ability to hold his mental tendencies in check, even though an active hallucinatory and delusional condition has existed for nearly four years, speaks in favor, perhaps, of a strong mental make-up."

In both Case I and II there is a chance for the patients to clear up, and clear up permanently. They both show insight and coöperation to a very considerable degree, enough to carry them through to health if we can have the time and conditions favorable for working with them. It must be realized that it is almost impossible to accomplish much in a busy clinic. Whether either of these cases do actually clear up or not, they belong to types that ought to be saved from ultimate deterioration. For this reason those interested in the urgent needs of these and allied conditions should exert their best efforts in behalf of the borderline psychiatric cases.

SERIOUS MALADJUSTMENT ON THE BASIS OF A COMPULSION NEUROSIS IN THE LIFE OF A SCHOOLBOY

CASE III; 1607; L. E.; age 18 years; schoolboy of Hebrew parents.

HISTORY.—*Family History.* Father died at the age of 56. Mother is living and well. She has six children all living and well. The patient knows nothing of his father's parents; his father has one brother and two sisters who are well as far as the patient can say. His mother's parents died when she was three months old; she has one brother who is healthy and one sister who is healthy. Among the cousins all are healthy as far as the patient can say. There are no indications of epilepsy, alcoholism, or nervous or mental disease in the family.

Previous History.—The patient does not remember having had any illnesses in childhood or subsequently. He gives no history of convulsions during childhood or later, or other nervous affection. He began school at six years of age and experienced no difficulties in school until the complaint for which he comes to the clinic, came upon him.

Present Complaint.—Onset. The patient first stated that the onset was four or five years ago but later could not remember exactly when it began. (Probably somewhere between the sixth and eighth year.) "I was in school, we were going to assembly, I touched the wall with my left side, then I said to myself, 'If I don't touch it with my right side I'll get appendicitis'. So, I went and touched it with my right side." (He means hip region.) "Then the trouble came on me; if I touched my face, I'd have to touch the whole of my face, or head. If I wouldn't touch it I would have to break (some accident would happen to) my head or neck. If I'd put my shoe on twice, I'd have to put it on a fourth time—a third time would not be enough. When I get up in the morning, I say, 'That shows I'll never be sick, that shows my father will never be sick, my mother will never be sick, my little sister will never be sick, that's the way it goes on for twelve different times'. (There are many varieties of this, for instance, 'That shows I'll never be run over', 'That shows that'll never fall on me, etc.') If I go by a hospital I will say 'I will never be sick because I will take care of myself, my father will never be sick because he will take care of himself, my mother, etc., etc'."

Physical Examination.—General physical examination negative. Neurological examination negative. Bowels normal, sleep good, appetite normal; does not dream.

Continuation of Present Illness.—"If I see a pair of shoes, one down the other up, I ought to fix them so that one is the same as the other; if I touch the wall (touches the wall of examining room) which is on the East side of the room I will go and touch the opposite, the West side of the room. I think that if I touch the East wall, unless I touch the wall on the West side, also, I will have to go and live on the dirty, rotten East Side. If I touch the lower part of a window-sash frame, I will touch the other three sides of the frame, and two sides of the frame above. Last night I went into a drug store, that's the second time I'd been there. I had to go again a third time as I do not like the number 3, I had still to go a fourth time; the impulse made me go. If I didn't go a fourth time I might have to go back for medicine for myself.

"In the morning when I get up I touch all my teeth and count two sixes, and two fours, and touch both sides of my face to include the whole head; I do it also when I break wind; I do it in the early morning so I have it to rely upon. I do it if I see something dirty or touch something dirty. (The patient got up to close the door of the examining room.) When I close the door I take hold of both knobs, and touch them both in the same way. (He touches the knobs alike, with the left and right hands, and the lock with his two thumbs.) What I do with one hand I must do with the other—I believe in equals.

"I have to go to synagogue tonight but I'm afraid to go there, as I have been there twice, and this will be the third time; I shall go but I have an impulse against going. At the seashore in summer I would go in bathing, and then a second duck. I'd swim on one side, then on the other keeping each side equal in swimming; I'd go in a third time and run right back again to make it a fourth. If I could get rid of that table nothing else would bother me. If I went in a fifth time I'd have to run out and go in a sixth; if I'd go in on the right side, I'd have to swim on the left to keep it equal. If I had to put my coat on a third time I'd have to put it on a fourth. If I see three sides of a box, I'd have to see the fourth; if

I'd see five lampposts, I'd walk until I'd see a sixth. If I'd take out three books from the library, I'd get a fourth.

"I always keep on counting, one, two, three, four, five, S. 'S' means a show, give the other five a show. If I'd touch one side of my coat, or stroke it, I'd stroke the other side also, and repeat the formula, stroking each side six times to make both sides equal. If I'd write three words on a line, I'd try to put in a fourth word, and it wouldn't always fit in, and I'd have to think and think; this has caused me to flunk in many an examination. If I rubbed out a word on the left of the page I'd have to rub out a word on the right, too; in fact, everything has got to be equal with me. Yesterday in writing an essay on milk, I made a mistake and spelled the word 'milk', *MLIK;* this word came on the extreme left of the page, so I placed it also on the extreme right of the page, and spelled it in the same way to make it even. Then I made 'M' six times, 'I' six times, 'L' six times, 'K' six times, on both sides of the page, to make it even.

"Then, I will cross my legs, say I cross the left over the right (left will be East), I will cross the right over the left, then back and forth, six times. The same with a handkerchief—if I have one in the right pocket, and one in the left, take the right one out, put it back, take the left one out, put it back, and so on, six times. That six law applies to everything; if I could only get rid of that. When I look at the page of a book, and see three letters, I look over the page to find six letters, and I strain my eyes doing this. Suppose I have three quarters in change, that's unlucky, so I will have to get one of them changed, I'd get two dimes and a nickel. If I cross my legs so that the left comes on top, I must do it back and forth six times to make it right. If the right leg happens to cross on top first, I do not need to do anything. If I have money in one pocket, and mother gave me some for errands, I'd put it in a separate pocket; if I'd forget I had money in two pockets, and put change in a third pocket, I'd have to put money in six pockets; if this happened again, and still a third time, I'd carry it up to six times, and then the whole formula all over again six times. If I lay my note book on the corner of the desk, I will touch the three other corners, to make it even, and then as one side of the book was laying against the desk, and there is an opposite side and four edges, I would go through the four corners, touch the four corners of the desk with the other side of the book, and the four edges making six." (Question, "Suppose you did not do this formula?") "Such thoughts would come into my mind!" "If I take five steps into a room, I'd walk back, and make it six steps, if I eat five rolls, I'd stuff down a sixth or else take only four." (The patient has regularly prefaced his story with, "I have told you, I think, all I have to tell you," and often interrupted with, "That's all.")

"When walking, if I saw three corners of a street I would walk back and take a look at the other corner. Putting on my collar, if it's a third time I have to make it a fourth, the same with my shoes, coat, etc., etc. If I'd eat a thing twice, and another week my mother gave it to me again I'd be afraid to eat it." (Question. I asked the patient what this third time meant to him.) "See, there's four in the family left, two married sisters, one married brother. Two unmarried sisters, my mother and myself are at home now. Three unmarried and my mother, that's four. If I touch this corner of the desk, there will be three corners left; immediately the impulse would come to my mind, 'If I don't touch the other three, there would be only three left in the family', so I touch the other three.

"No. 1 means my father; No. 2 means my mother; No. 3 means my family; No. 4 is myself; No. 5 is my little sister.

"If I would be walking on the street and happened to turn round to the left, and see—say, a subway station, two sides of it, I'd have to turn to the right. But if I saw it on my right side, first, then to make it even I'd turn to the left—I would not leave it on the left, but go through the whole formula. I'd want to make it come out on the right side. I'm compelled to go through the whole table. I'd say, why not give the others a show? I could not stop at four, for instance, for why not give my little sister a show, one, one, two, two, three, three, four, four, five, five, S, S, for show. In school, if the teacher would be explaining something I may have to do one of these formulas, and it would distract my attention and I would not be able to follow. (Question, "Suppose you did not do the formula?") "If it was on the right side, I'd say, I'll go crazy on that side; if I read three paragraphs in the newspaper, and didn't want to read any more, I'd have to finish it and go through the formula." (Question, "What would happen if you didn't?") "I might die. If I'd see three wheels of an ambulance, I'd make a dash to see the fourth; otherwise, I'd be run over by that wheel if I didn't see it. If I'd see a douche tube, and touch it with my right shoulder, I'd have to touch it with my left, otherwise I might have to have a rectal injection. Suppose I happened to touch liniment with my left hand, I'd have to touch it with my right, else I might sprain my right arm. Running for a train, suppose I take five double steps, I must take six, or I'd die, or be killed by the train. I have missed the train many times. I am surprised I wasn't run over. If I touched this window, and didn't touch that, I'd say, I'd fall out of that window if I didn't touch it. Almonds are three for a penny; I wouldn't eat three—I'd throw one away, or eat two and a half. If I'd eat it, I'm liable to get appendicitis or something. I just now looked at my left foot, and said to myself, 'If I don't look at my right foot, something will happen to it'. Suppose I lay down in bed three times; if I don't lay down a fourth time, I am liable to get sick and have to lie down. The thing is, if I don't do these things something will happen to me—death, sickness, accident, go crazy, go blind, deaf, dumb, anything you can imagine. If I touch my left eye, and not the right, I'd say, 'I'll go blind in that eye'; the least little thing I do there's an impulse." (I asked the question again, "What would happen if you didn't do these things?") "God—there's no end to that! If I'd pass out of the door turning to the right I'd say, 'Why not pass the left side, too', and say to myself, 'If I don't pass on that side, I may go crazy on that side'. If I walked on five stones, I'd go back and go on six stones. If I didn't, five means death. Yesterday I took a plate of rice, the fifth within a week; I did not eat it and left it." ("What would happen if you had eaten it?") "Five means death. I have my left leg against the desk; if I didn't get up and touch the desk with my right leg something would happen to the right leg—break it, fall. The left shoe-string dropped to the floor upon lacing my shoes this morning. I must unlace the right one, also, and allow it to drop to the floor, repeating both six times." (Question, "What would happen if you didn't do it?") "I'd go crazy on that side. Sometimes I get pains in various parts of the body if I do not do the formula—pain in the head, stomach, etc., etc., etc." (The patient had to leave the seance for attendance at the synagogue. I said to him, "Suppose you didn't go?") "When your father dies you have to say Kadish three times a day for eleven months." (The above was given in two or three separate sessions.)

I asked the patient to tell me all he could remember about the beginning of his trouble. He is indefinite about it; at one time he says it began suddenly, at another, he isn't sure.

"I don't remember it being as bad as it is now; it gradually gained on me. When company was home, I never did it; now I try my best but the impulse is too strong. I can't avoid doing these things. I thought I'd be able to force it away but saw it was gradually gaining on me. I don't remember doing it in my childhood, in fact, I don't remember at all when that impulse came on me. Since my father's death (four months ago) it has been stronger. Since his death all these impulses are non-resistable."

After considerable questioning, and urging, the patient was absolutely unable to remember anything that led up to the onset. He repeats over and over that the onset was sudden.

" I remember that my father once told me that in Europe when a man breaks wind he washes his hands with water from a bottle of water which he carries in his pocket. This led me to the idea to do that, that gave me the idea to count, not by means of the table but just counting right along, and that led me to say I'll give my family a show, and I made that table. The table led up to 'that shows—'" (Question, "How do you mean?") "If I saw an accident, for instance, I'd say, 'I hope that never happens to me', then I applied the table, 'I hope that that will never happen to my father, to my mother', etc., etc., etc." (Question, "What do you mean by 'That shows—' ") "I might say 'That shows' instead of 'I hope'. 'That shows' is stronger than 'I hope' and I wanted to make it more definite and certain."

(In the above the patient tries to trace, and explain, the development of his compulsion, but without success. He merely becomes involved.)

"The washing of hands led to the morning practice I spoke of—touching the face, and every tooth. Before I took up the table I would say numbers without limit, but never stopping on an odd number, I would keep on saying them the whole day. I just got the idea. I was always afraid of an accident, and I said to myself, 'If I say that, that won't happen to me' and I still believe it today. I didn't want it to happen to me and thought that would protect me; there are millions of things that might happen to me."

Repeated efforts to force his memory back to the beginnings of his compulsion, and ideas related to it, fail absolutely; he is unable to state anything definitely, and contradicts himself over and over as to the time when his compulsion began, and refers it variously to different episodes. It is expected that he must fail at this.

I have seen this patient at fairly regular intervals with the exception of a three weeks' intermission when he stayed away and when I was not sure that he would ever return. It has been my purpose at each interview to allow the patient to speak out that which is in his own mind without suggestion from me. This is, of course, the regular analytic technique, or at least one small item in it. As I proceed with him I expect to find that I am working against the continual, unconscious resistances of the patient against bringing his conflicts into view.

I shall, I think, proceed with the history by taking up the development of his own unfolding conflict as the sessions progressed. In the course of his talk in one of the first sessions he speaks of heart failure which has caused him a great deal of worry.

"I test my heart—take out my watch; I think the cause of heart-failure is if I did anything like that, ran too much; sometimes I can't do without anything in my hand; whenever I sit idle I don't know why but I do it, take this pencil and hold it in my hand, write something. In the middle of my work I get down and play with the dog. . . . Masturbation comes to my mind. I finally broke away from that; for a month or two I kept it up and then broke away. It tries to come back on me and that's the reason I get drowsy and sleepy." This suggests a reference to hand-washing which he has previously given.

"This drowsiness prevents me from studying at times; it takes up a lot of my time. I have to stop work and go out and have a game of basket-ball or talk with the folks. It's about one and a half years since I masturbated. I told you I continued it for a couple of months, it was eight months. I wanted to ask you how to break away from that desire." (Question, "Have you ever connected this with your nervousness in any way?") "No. Of course, I had an idea we were going to lead up to it. I want to get my mind away from that—what can I do? Some of the boys said to go to a woman; I heard it weakens one to do this. I heard, also, that auto-intoxication comes from masturbation. It's a bore on your mind and hard to resist. It makes you feel drowsy, sleepy, dirty, nervous. It causes serious illness like auto-intoxication—whatever that is. Does it cause craziness? It has something to do with the mind I know."

In the above the patient is able to set aside a little resistance and speak of the conflict of masturbation. I have always instructed him to continue to speak whatever comes into his mind no matter to what it refers, or how silly or unimportant it may appear to be. It is the production of his own mind which we are to study.

"When I am at home, alone with my mother I start touching my mother's shoes." (The fear of his father's criticism, and the impulse to touch his mother's shoes will not be clear just at present but I think will explain itself a little later on. Perhaps I had better say here at least there is an affect of some sort directed toward the mother, and a fear of some sort which relates to the father. This is the first reference, also, I believe, to a relation between the love-impulse and his compulsion.) "She takes a broom and slams it across my hands. The death of my father added a lot to my impulse, I think." (It is important to note this relationship of the patient to his father and mother. It is the key to his whole history. Here he brings it up spontaneously in connection with a dream that he brought me. He does not know to what depth it will take him; if he did, that is the last that I should see of him.) The patient continues, "Father was very unselfish, he cared for the wants of his family. He cared for us, not for himself; he looked weak and ill. When he was ill I said to him, 'There you go, you want to go out again, you were just ill'. When I go to bed I have a habit of thinking how he was lying in the coffin; I try to shun these thoughts and keep them away from me." (In these thoughts of sorrow for the father which as we shall see are excessive, we might suspect an over-compensation for previous uncharitable thoughts. An incident happened one day as he was leaving me showing this relation of the father as transferred to me. He said as an expression of his appreciation for the trouble I was taking with him, "I feel I ought to repent you." I called his attention to this error at once, and he said he meant "repay." But I know that his attitude of repentance as I have indicated is because of a failure to repay his father; that is, take the aggressive against him, at times when he felt the justified impulse to do so. So I, also, shall have to be on guard for these same impulses toward me. This conception will fit in also with his fear of the father's criticism. Having this in mind I ask him the question, "Do you remember ever treating your father badly?" He replies, "I don't remember being really bad to my father." But we shall hear more about this, whether he was really "bad" to his father, or had evil thoughts against him of a violent nature which he did not dare to express. It will become plain before I am through, that his father stood in the way of the expression of his intense love-impulses toward his mother.) "I hardly had an argument with father; I couldn't talk back to him he was so kind-hearted." (Here I might dare to hope that anyone who takes the trouble to read this history will, after they have become better acquainted with the father, turn back and re-read this passage.) "I think the cause of my badness is (the patient is here speaking of some impulses which he has at home of an irritable character such as, "I'm a big crank about eating," "I get sore when things are not just as I want them, and perhaps I'll cry out, 'Some breakfast!' 'I shouldn't do this. I'll say to myself, '. . . let's tease her a bit', and I'll think of things she doesn't like to have done to her; then I'll feel sorry I did it; I lose my temper and won't think to control it) that I don't want to give in." (Again the patient is more right than he knows.) "I did not appreciate father's great kindness when he was alive and it makes me burst with shame. I antagonized and aggravated him a good deal—I regret these things."

The patient also thinks that his father might have lived longer if he had treated him more kindly. He might have stayed in the house instead of going out and exposing himself to bad weather. So much for the reactionary impulses which the patient felt but which he did not dare to express against his father, as brought out in this sitting.

At another time the patient told me a little of his association with boys and girls, making a few general remarks such as, "This girl is very attractive and that one is silly," in speaking of two girls who used bad language, one of whom, so he thought, was of loose moral character, liked to dress too much, and painted. One of his boy friends "fell" for them. He said that they thought they could get him (the patient) for a boob; he dropped them. The patient then told me, "In my mind an impulse says, 'Tell him anything', lie to him, even." "I liked one girl but she was kind of tough. (A little of this will show something of the patient's environmental temptations.) She paints and powders. Once she said, 'I charge five'. 'Oh', I said, 'Is that all—for what?' and kidded her along. Another girl was in a certain store, she was talking along; she talked smutty; I wouldn't touch her; she's too dirty. I have more enjoyment seeing the thing than doing it." The patient also speaks of another girl with whom he had a lot of smutty conversation, and of still others whom he knows are loose. These instances, and many others, represent the patient's opportunities for sexual experience, which he did not gratify in any way, and against which he was struggling.

"I feel very bad; this impulse (compulsion) gets worse at night; these things we talk bring back the old habits again; the six habit was going; everything might be washed off little by little; now I touch things for ten minutes and lose time all day long." (All this is nothing but the patient's defenses and resistances against analysis which I realize is very serious.)

He told me later that when he was a child, the family was crowded into a few rooms—he slept in the same room with his father, mother, and little sister, and frequently in the same bed.

At each visit the patient betrayed continuing anxiety over his condition, and has asked over and over again if this treatment is going to get him well—and how it was going to cure him. His mother and others complained that he was getting no better; what, therefore, was the analysis good for; wasn't he taking any medicine (I had given him a prescription which he had refused to take, stating that he did not like to take medicine although he had asked for the prescription). His nervousness was increasing; he showed plain signs of the doubt in his mind as to whether or not he should go on with the analysis; on certain visits his compulsion was so bad that I myself felt very uncertain whether I should be able to hold him. At one visit he complained that instead of getting better he was getting worse. "I do it now more than I ever did; I don't see how I am going to get rid of it in this way; I thought I'd get some relief by this time; I have been coming here for a month; I don't know what to say except to emphasize that I am getting worse. I don't think the mind can ever be studied . . . I'm afraid, I don't want to get nervous, I'm afraid of a nervous breakdown; when I am alone I can't find myself, my nerves are so unstrung; if it will come to the worst, I'll force it out myself. (He intends to say he will stop it by supreme effort of will.) I have enough power in myself to do it, I'll bandage my hands for a month; I'll chop off my arms; that'll be a month of hardship, that's all! Instead of six, I do it thirty-six times now! I'm afraid to sleep alone." (The patient is full of resentment and resistance today. His mood is irritable and his expression is a sneer.)

I did not see the patient after this visit for nearly three weeks, and I began to think the sessions were ended. When he appeared again, however, it was of his own accord, but before I began with him the work of the analysis again, I allowed him to visit me several times without resuming the analysis, wishing to be sure that he had a keen desire to continue. During his absence he had consulted one or two physicians about his case, as well as others, who advised him in various ways and sneered at the work that he had been doing with me. All this was in line with the doubts and anxieties which were filling his mind during the previous visits, already mentioned. On returning, the patient asked me again concerning this analysis. In what way would it cure him? I remind him that while I might allow myself to engage in discussion along this line with him, if I did so I should fail to get what we were after, the productions of his own mind, not mine; he might have a subconscious purpose in launching such a discussion, that of keeping back real issues. He then admitted there were some things he kept back and did not tell me. For instance he recalls seeing his mother and father in the act of coitus. He slept in the same bed with his parents and his recollections of these occurrences go back to his fourth year when his sexual curiosity first became aroused.

I shall here conclude this introductory report of the case. There are many interesting points in it. The patient is practically analyzing himself; he consciously or subconsciously, bit by bit, discloses everything. The patient interprets for us, at least partially, the meaning of his 1, 2, 3, 4, 5, 6 formula. He has spoken mostly of his mother who is No. 2. His emotional relation to his mother is now clear. No. 1, "the superior" he has spoken of several times in his story, but he had deferred much reference to No. 1 who is, of course, his father. There will be more to be heard about No. 1. He has spoken also of the intensity of his conflict against gratifying his impulses and he has told me of the intensity of his conflict against No. 4 in getting away from the masturbation. There is such an abundance of material in this case that it would alone fill a book.

How No. 3 finally comes into the formula is partially seen but as there are sisters and a brother in the family, No. 3 includes them also; they do not appear in the story yet, but they come into it later on. No. 5 refers to one of the sisters—in what way we must leave for the present. No. 6 he has himself disclosed in both conscious and automatic reference to "six times six." Also the text says "and six for a show, to give everybody a show," that is, to make it even.

In this case we learn something more about childhood than we like to believe can exist. I might add that this boy's disorder gives us an insight into the possibilities concerning the genesis of other forms of the psychoneuroses.

This case indicates even in such interpretation as I have been able to give, the possibilities of amelioration for these unfortunate patients. It suggests also that there is plenty of room for work in preventive medicine and the social service departments, and illustrates, perhaps, that the earlier we can get hold of the psychoneuroses and the borderline cases, the more likely are we to avert the precipitation of an actual psychosis.

A CRITICAL REVIEW OF THE PATHOGENESIS OF DEMENTIA PRECOX IN ITS RELATION TO THE BORDERLINE PSYCHOSES

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There are few medical problems more hopelessly tangled than are those which have to do with our conception of the mechanisms of the so-called functional psychoses. One searches the literature in vain for evidences of a common ground upon which various unquestioned authorities in psychiatric matters meet. Typical of the whole matter are the widely divergent views of many alienists concerning the conception of the nature of dementia precox. For example, in a symposium published by Drs. Jelliffe, Meyer, and Hoch on dementia precox,[1] Dr. Meyer, in discussing the lack of harmony of the various conceptions of dementia precox, states: "That quite recently Alzheimer reemphasizes the adherence of the nosological orthodoxy to grouping dementia precox with the essentially organic diseases, and not merely as what I would call an incidentally organic disease." This statement of widely diverging views of two authorities summarizes the situation, not only as regards dementia precox, but also all the other psychoses, the pathological basis for which is unknown or poorly understood. Indeed, it might also epitomize the state of feeling that exists concerning some other functional conditions, hysteria, for instance, and many of the neuroses and anxiety states. Here, too, there has been an increasing tendency on the part of some to explain the symptomatology of these conditions by reference to disturbances, not only in the affective and intellectual life, but also to abnormalities at physiological levels, mainly in the glands of internal secretion alone or together with disturbances of the sympathetic and vagus systems.

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[1] Jelliffe, Meyer, and Hoch. 1911, Dementia Precox, Badger, Boston.

I am making reference specifically to dementia precox in this discussion of the borderline psychoses for a number of reasons: first, because dementia precox is by far the most common type of psychosis in actual percentage of occurrence; secondly, because dementia precox-like reactions are very frequent without the precipitation of an actual psychosis, and thirdly, because if we can arrive at any conclusions concerning dementia precox as regards its organic causative basis, then we will be enabled to draw corollaries which will be of use in measuring our knowledge of other so-called functional conditions. It seems to us that the crux of the whole matter is still that timeworn psychological problem. Is it possible to have mental manifestations which are abnormal occurring without structural changes, not necessarily in the brain alone, but either primarily in the brain or secondarily? Is it possible to answer this question at present? We shall make reference to the writings of Jelliffe, Hoch, and Meyer, who, it seems, have taken the stand that dementia precox is a functional disease and that it is essentially a *disorder of the personality.*

They seem to agree that such organic changes as occur, either in the brain or in the other organs of the body, must necessarily be considered as secondary associative processes to the primary character change. As adherents of the view that dementia precox is an organic disease, either of the brain, of the endocrinous glands, or some other organs, the brain changes being secondary to these, we will refer to Southard, Alzheimer and Rosanoff.

In discussing Meyer's view of this subject, we must first of all give his definition of dementia precox. We find that he characterizes the dementia precox group as those "types of defect and deterioration which show the existence of the development of fundamental discrepancies between thought and reaction, defects of interest and affectivity with oddities; dreamy, fantastic or hysteroid or psychasthenoid reactions, with a feeling of being forced, of peculiar unnatural interference with thought, frequently with paranoid, catatonic, or scattered tantrums or episodes." [2]

Being an exponent of the theory that dementia precox is a functional psychosis and that, therefore, it occurs independently of any pathological changes in the body, including the brain, Meyer divides the activities of the individual into the mental activities or reactions which occur as a result of more or less conscious association, and the non-mental nervous functions which are those that can be produced by electric or reflex stimulation. These may occur with or without consciousness. The non-nervous functions would include the various activities at physiological and psychochemical levels. He says, "Psychogenic disorders are those which depend on conditions or events which can only be described satisfactorily in terms of psychobiology; actions, emotional reactions and attitudes, and intellectual or 'thought' constellations, and their conflicts and abnormal combinations, or abnormal reactions, with their effect on the general mental balance." These psychogenic elements occurring in conflict combinations produce mental states of various types, for instance, hysterias, amnesias, various "substitutive activities," and, in some cases, produce actual psychosis. Secondarily, disturbances occur which are not represented as being part of the actual personality of the individual, such as those conditions which the author calls submental disorders seen in "tremors, nervous dyspepsia, fits, contractures, vasomotor changes, metabolism changes, etc., or they may even simulate focal diseases of the nervous system (hemiplegia, and so forth)."

Meyer seems to believe that these submental abnormalities are actually connected with the trouble by means of a more or less conscious association and that the important factor in the production of all types of psychoses, excepting the organic, are not the secondary submental or non-mental changes but the primary disorders of consciousness. He describes three classes of psychotic conditions which must be considered: first, those ordinarily termed functional psychoses which he says are due to collisions of function with incidental disorders of the organic balance of these functions, citing hysteria and psychasthenia as examples; second, consideration must be given to the evidences of submental malfunctions such as toxemias or metabolic changes which occur, for instance, in alcoholism, syphilitic processes, hyper- and hypothyroidism; and third, reference also must be made and proper value given to symptom complexes produced by focal lesions in the brain, such as tumors, and so forth. With very clear vision, Meyer sees that it will be possible for us to arrive at definite conclusions concerning a given psychosis only by careful correlation of the data received from all three lines of investigation. Then he immediately proceeds to forget all of them in the discussion of dementia precox, except the first. It may not be out of place here to say that it is the writer's opinion that the reason why psychiatry has failed so signally to keep apace with the advances made in other lines of medical endeavor is the undeniable fact that psychiatrists have not always studied their material in the broadest way. The psychiatrist who wishes to arrive at the truth about his cases must be carefully trained, not only in normal and abnormal psychology, but equally well versed in the technique and application of the science of neuropathology. Extremely important, of course, is a knowledge of normal anatomy, both gross and histological. Nothing will ever be contributed to the subject by descriptive studies, no matter how keen or well done. Meyer dismisses the neuropathological evidence in dementia precox as being meager and refers particularly to the work of Kleist. He says, "the isolated facts of the frontocerebellar disorders, tremors, reflex alterations, dermatographia, seborrhœa, and the eye symptoms appear like elements in the general process but not like helps for an explanation." Referring to the histological data, chemical findings, and the work of correlation of the clinical symptoms in dementia precox with the organic changes found in the brain, the author dismisses these as being merely incidental or due to defective oxidation or possibly the consequence instead of the cause of the symptoms. Meyer follows Freud and Jung and emphasizes the psychopathological viewpoint, ascribing the difficulty in this condition to habit deteriorations and tantrums which he says are pathologically unfavorable to the maintenance of a normal mental balance. He speaks of complexes, habit conflicts and all the other psycho-analytic mechanisms as the essential causative factors. The symptoms of the disease, he says, are due to the peculiar attempts which have been made at balance and reconstruction. He admits, however, that undoubtedly a large number of cases are "beyond complete analysis and understanding." How exasperatingly vague this viewpoint is can be seen from a statement of what he regards as the general factors of the dementia precox course, "the oddity, unnaturalness, and incongruity of the entire picture, in the face of relative clearness, and the tendency to turn on definite complexes, and these especially in spheres which are difficult to reach for an adjustment." To the author this is typical of the enigmatical statements which so frequently occur in the writings of those who wish to apply psycho-analytic principles to the study of this disease.

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[2] Meyer. 1908, *Psychiat. Bull.*, v. p. 257.

In discussing the situation, Meyer recognizes the fact that he must answer two arguments which "are raised against the dynamic importance of the psychogenic material." These objections, of course, are the actual deterioration which exists mentally in these patients, together with the undoubted definite lesions in the brain which are almost invariably found at autopsy. Without considering either of these points, he dismisses the situation by saying, "How can we account for the stabilization on a lower mental level, and what is the relation of the degenerative histological processes observed in many acute cases?" The answer may be illuminating, but I confess that it is not helpful to me. Kræpelin in his theory of the causation of this disease or, for that matter, manic depressive insanity, is just as indefinite as Meyer in discussing his psychobiological conception of dementia precox. Kræpelin also talks evasively of disorders of metabolism, toxemias, poisons of various kinds, and so forth, and more latterly of imbalance in the glands of internal secretion.

While Jelliffe in the same monograph is

inclined to pay more respectful attention to the pathological evidence submitted as the causative factor of dementia precox, he spoils it by saying, "I freely admit that we are still much at sea in this matter and am fully prepared to follow Meyer in part in a more functional interpretation of certain of the dementia precox reactions." In attempting to recognize a fundamental personality he is "directly opposed to a too dogmatic pathological interpretation. After all, our pathological findings may represent atrophies of unused association tracts which have resulted from the, so to speak, petrification of bad habits of mental adjustment." This is exactly what we believe Meyer meant to convey as his conception of dementia precox. While Jelliffe very nearly committed himself when he compared the changes in dementia precox with those found in general paresis making the diffuse character and slow progress specific for dementia precox, he would not have been true to his extreme devotion to psychoanalytic principles if he had not finished up with the statement just quoted. Nevertheless, it is encouraging to hear him say, "So that to the more striking mental signs are added physical signs of almost as definite a character as those met with in paresis. . . . . The clinical pictures are bound to begin in a slightly different manner, according to the anatomical localization of the processes. The course will vary by reason of the same factors of variability in contact of the pathological alteration, and the general end level will be reached largely as in paresis by the more or less general diffusion of the processes in the areas of special predilection."

Hoch [3] in discussing the pathological findings in dementia precox says, "While these findings, upon which rests the claim that dementia precox is an organic disorder in the same sense as is general paresis, cannot be neglected, and represent a most important field for research, there is another set of data furnished by an analysis of the constitutional factors in these cases, of the development of the symptoms—data which would seem to show that, granted all the findings of an anatomical and, perhaps, chemical nature, dementia precox is after all not a condition which can be placed side by side with the plainly organic diseases, such as general paralysis."

"And so, after a discussion of the differences between the organic disorders accompanied by dementia and the nature of the dementia in schizophrenia, he veers toward the viewpoint that the important thing to be considered is the personality of the individual as represented by psychopathological considerations. Indeed, says Hoch, "This is something which these cases of dementia precox have in common with some disorders such as hysteria, certain simple paranoic states and some psychoses of degenerates, that is, conditions in which the psychogenic nature of the symptoms is now scarcely questioned by anyone." He recognizes also that there is a close relationship between the conditions mentioned which he states are undoubtedly psychogenic in nature, and dementia precox. He then goes on to describe the peculiar type of make-up found in the personality of the patient who suffers from dementia precox. He lays great stress on the fact that peculiarities of make-up are found in dementia precox and that the development of the disease is a direct outgrowth of these peculiarities.

When we come to find evidences upon which the matter of personality and its influence in the development of dementia precox is based, we see that Hoch studied first, in 1908, seventy-two cases and found his typical "shut-in" personality in 35 per cent. whereas in 16 per cent. it was "indicated," so that there was some evidence of it in 51 per cent. of the cases. He refers to these as striking figures because they were taken from cases in which no particular attention was paid in the history-taking to matters of personality. However, in his more recent material amounting to thirty-eight cases he found a typical "shut-in" personality in 49 per cent. and an "indication" of it in a further 19 per cent., making a total of 68 per cent. altogether. Besides the typical seclusive, shut-in personality, 24 per cent. of these 68 per cent. showed other peculiarities of make-up.

Kirby of the Psychiatric Institute studied 100 cases of dementia precox and found a plainly shut-in personality in only 50 per cent. of the cases. When one considers that these personality studies were made with a view to bringing out a definite defect it seems hard to understand why it is necessary to commit oneself so definitely to the statement that dementia precox is a perversion of the personality. What happens if one begins to question this theory by referring to the other 50 per cent. which did not have the typical seclusive or shut-in personality? In order to make reasonable the psychogenic viewpoint, this question is answered by Farrar[4] who recognizes five types of children having peculiar personalities which are particularly apt to develop dementia precox. Despite the fact that these groups are widely inclusive and constitute classes in which every child living may be placed, the matter, for him, is definitely closed, and so it is conclusively proven that dementia precox occurs on a groundwork of defective personality which is the dominating feature of the development of this disease. These five groups are as follows: backward types; precocious types; neurotic types; asocial types; and juvenile types. The ingenious attempt made at all-inclusiveness is apparent when a discussion of this type is given. Farrar says that the history of these "juvenile types" usually show "that they were bright enough and made progress in school, at least in certain subjects, and yet their mental growth was not normal. The impression that they made as they grew older was that the development of the maturity-reaction was not keeping pace with the years." To be fair to the author, however, who has really written a splendid article, we must quote this statement: "There are no sharp lines of demarcation. The types are necessarily more or less arbitrary. . . . They deal mostly with observational data, and while we can doubtless derive from these data certain clues as to the correlation and succession of symptoms, they do not as yet offer any suggestion of explanation for the individual or type-anlage as such."

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[3] Hoch. *Proc. Am. Med.-Psychol. Ass.*, xvii, p. 227.

From whatever observations I may occasionally have made, it must be already evident that I have shown considerable preference for the side of the argument which leans towards the conception that dementia precox is an organic disease of the brain. After we give the organic side of this question a small chance for a hearing, I shall refer to a fair discussion of the whole situation recently made by Dr. Kirby which probably covers the present status of this subject.

The work of Southard[5] cannot be dismissed without serious consideration. So important is it that I have considered it essential that eleven of his conclusions be copied verbatim.

1. Existent evidence for the organic nature of dementia precox is not wholly convincing, since (*a*) the cytological changes described are found also in cases of toxic deliria and in cases complicated by severe visceral disease and (*b*) the stratigraphic changes described are found also in certain senile cases without characteristic symptoms of dementia precox.

2. Resort must, therefore, be had to the topographic idea, for the adequate exploitation of which total-brain sections, with cytological exploration of all areas, are ideally necessary.

3. Random blocks of brain tissue with demonstration of satellitosis, infrastellate gliosis, or disintegration products of cell disorder will throw little light on the mechanism of dementia precox.

4. The data of the functionalists (dissociation, sejunction, intrapsychic ataxia, and the like) are of the utmost importance as indicating the essential focality of the pathogenic process and the focal variations in its severity.

5. The curability of certain cases, the remissive character of some cases, the speedy disappearance of particular symptoms, the persistent complexity of reaction in some instances, the absence of characteristic severe projection-system symptoms, all indicate that the process is histopathologically mild and that the focal changes found will be but slightly destructive or even irritative (in the sense of slight injuries readily repaired or compensated for).

6. Grossly destructive lesions of a transcortical character in Wernicke's sense might conceivably effect, for example, a permanent catatonic complex and doubtless will be found to do so occasionally; but the protean and progressive character of dementia precox will exclude such transcortical injuries from playing a large part in the pathogenesis.

7. The focal lesions to be sought for will doubtless escape macroscopic notice in many instances, since the volume of apparatus engaged in effecting very prominent symptoms is often slight and spread very thin in numerous areas.

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[4] FARRAR. 1916, *Psychiat. Bull.*, July, p. 323.

[5] SOUTHARD. *Proc. Am. Med.-Psychol. Ass.*, xvii, p. 261.

8. Studies of the "soft brain" and of gliosis in epilepsy have proved, however, that even comparatively slight degrees of cortical gliosis can often be palpated at autopsy.

9. Palpable glioses of a focal or variable character, combined in numerous instances with visible atrophy and microgyria, have been found in over half the series under examination, in cases regarded as clinically above reproach and *not* subject to coarse wasting processes, focal encephalomalacia, cortical arteriosclerosis, or diffuse chronic pial changes.

10. The frequent co-existence of several foci of sclerosis or atrophy in the same brain and the microscopic observation of milder degrees of nerve-cell disorder and gliosis in regions without gross lesions tend to the conception that the agent is more general and diffuse in its action than would seem at first sight, so that future research may well demonstrate that certain instances of coarse brain wasting and even of diffuse chronic leptomeningitis belong in the group (microscopic corroboration necessary for assigning values to focal variations).

11. The microscopic examination of the residue of cases in which gross lesions or anomalies were not described shows the same tendency to gliosis and satellitosis in numerous instances and the same tendency to focal variations from gyrus to gyrus exhibited by the gross lesion group. These findings suggest that the minor gross lesions and anomalies of several cases actually escaped notice (the protocols, though drawn up with a certain system, are by various hands) at autopsy, so that the probable actual proportion of gross lesions is sixty-eight per cent. If microscopic evidence is resorted to, the "organic" proportion in our series rises to eighty-six per cent.

Southard in the same volume also quotes the work of Sioli[6] who found in all parts examined of twenty cases of dementia precox certain evidences of destruction of nerve tissue, and then goes on to describe the character of these changes, which were mainly disturbances of the stratigraphic arrangement of nerve cells. He found degenerative products in spaces around the blood vessels and other substances were found in the tissues which were stainable by certain methods. Adventitial cells were also found *en route* to the vessels. There was gliosis, particularly in the subpial zone, deep cortex layers and in the white matter. Amœboid glia cells were also found in the white matter. Southard refers to the work of Alzheimer[7] which he has abstracted and quoted. Alzheimer says that a great deal of light has been thrown by his methods of staining on the pathological conditions found in epilepsy, deliria, and catatonic excitements. He quotes the distinguished neuropathologist as saying that dementia precox and epilepsy are severe organic diseases. Alzheimer has gone a great deal further than Southard and has even risked differentiating histologically "between a catatonic excitement and a mania, because the catatonic case will show characteristic amœboid glia cells, whereas the mania will not." Southard criticises this statement and points to the fact that deliria and catatonic excitements will exhibit similar changes.

Rosanoff[8] in his study of the "index of atrophy" using as his material autopsied cases of all classes of dementia at the Kings Park State Hospital and the Worcester State Hospital in Massachusetts, came to the conclusion that "the data show with striking consistency that mental deterioration of whatever nature goes hand in hand with brain atrophy. That this should be true in respect to arteriosclerotic dementia, general paresis, and senile psychoses might indeed have been expected in the light of previous knowledge of these conditions; but the remarkable fact is that it appears to be equally true in respect to dementia precox."

The material studied consisted of 452 cases. Realizing the fact that this was an important finding, Rosanoff very carefully went over the material as it had particular reference to dementia precox and came to the conclusion that "dementia precox is associated in some way with changes in the brain which lead to atrophy." It would not be apropos in a paper of this kind to go much further into this question. I will, therefore, take the liberty of referring to the views of Dr. George H. Kirby of the Psychiatric Institute as representing the opinion of a man who is willing to examine the situation from a coldly critical point of view, ready to accept facts when confirmed and unwilling to surrender individual concepts without important reason. Having been originally trained by Meyer and Hoch, the conservative views of Kirby are worthy of careful attention.

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[6] SIOLI. 1909, Histologische Befunde bei Dementia precox. 82. ordentl. Generalvers. des psychiat. ver. du Rheinprovinz am 14 Nov., 1908, in Bonn. *Zentralbl. f. Nervenh. u. Psychiat.*, xxxii, 1909.

[7] ALZHEIMER. 1910, Beiträge zur Kenntnis der pathologischen Neuroglia und ihrer Beziehungen zu den Abbavvorgängen im Nervengewebe. Nissl u. Alzheimer's Histol. u. Histopatholog. Arbeiten, 3, 3, 1910.

[8] *N. Y. State Hosp. Bull.*, ii, p. 200, 1914.

In reviewing Kræpelin's latest work, Kirby [9] finds that the author expresses the firm conviction that in dementia precox we have to do with a widespread and severe disease of the cerebral cortex. Kræpelin refers to the work of Alzheimer and Nissl, who found marked lesions in the second and third layers of the cortex with sclerotic nerve cells, infiltration of cells with a lymphoid substance, disappearance of nerve elements, overgrowth of neuroglia and appearance of amœboid glia cells. Kræpelin tries to correlate these anatomical findings with the clinical expressions of dementia precox. Kirby says regarding this attempt, "This discussion impresses one as being highly speculative and is not in the least supported, as far as I know, by any established facts regarding the physiology of the different parts of the cerebral cortex."

Kirby, however, gives fair consideration to the findings of Kræpelin who thinks that the fact that the frontal cortex is found to be most severely affected would correspond to the loss of those "higher and finer mental qualities which are generally accredited to the frontal lobes" as against the preservation of memory and knowledge which has already been acquired, and which forms the distinctive dissociation so characteristic of this disease. Very properly Kirby asks why, if as Kræpelin says, there is a correlation between the disturbance of volitional impulses and voluntary movements with damage to the anterior central convolutions, does not such damage lead to actual motor paralysis or apraxia? Here it may be stated, however, that a careful perusal of Southard's material will show that a great many cases which form the basis for his study referred to above showed clinically definite disturbances of speech and motion ranging from complete mutism to dyspraxia and apraxia. In passing, it may be well to call attention to the fact that Kræpelin recognizes a large subgroup of dementia precox cases to which he gives the name of schizophasia. In these cases Kræpelin definitely describes various aphasic, paraphasic, and apraxic and dyspraxic phenomena. Kirby continues by calling attention to the fact that Kræpelin ascribes the predominance of auditory hallucinations in dementia precox to points of irritation in the temporal lobe. The latter also postulates a theory from the findings in which the second and third cortical layers are most involved. He ascribes to these layers the functions of elaboration or translation of perceptions into concepts and of sensations into feelings and of impulses into activity of the will. In this way he explains the schizophrenic symptomatology and holds that if Kræpelin's theories based upon Alzheimer's findings are confirmed, then disease of the small cell layers of the cortex "must be looked upon as being responsible for the mental disturbances characteristic of dementia precox." Kræpelin explains the preservation of memory and acquired knowledge on the theory that these have their seat in the deeper layers of the cortex which are not involved in this disease. Kirby continues, "We cannot, under any circumstances, dismiss lightly the views of one who has exercised the greatest influence on modern psychiatry." He further states, "Widely divergent views are held regarding the nature of the disease; as to its causation nothing definite is known, and by at least one group of psychiatrists it is more and more questioned if what we call dementia precox is after all to be looked upon as a disease in the usual sense of the word." Here is a very fair statement of the facts but very clearly can be seen the influence of the teachings of his predecessors on the mind of this clear psychiatric thinker.

All this discussion is in my opinion extremely important as an introduction to a study of the borderline psychoses. The doubts and uncertainties which enshroud the problems connected with dementia precox hold good, not only for the actual psychoses which are considered functional such as manic depressive insanity, paranoia, paraphrenia, and so forth, but also exist in the discussion of the pathogenesis of psychasthenic states, hysteria, compulsion neurosis, and even of the ordinary neuroses. It is hard to see how even the study of the problems connected with neurotic, backward and defective and criminal abnormal types can escape inclusion in this discussion. The truth of the matter is that we do not know definitely the pathogenesis of any of these conditions. It is easily conceivable, therefore, that a discussion of borderline psychoses may lead to a great many indefinite statements because we are starting from a very uncertain premise. First of all, the so-called normal types are but little understood. It is unfortunate that a fairly thorough review of the literature finds it barren of any definite information concerning what is to be considered the normal mental reaction type. It is well, therefore, to ask what are the borderline psychoses? The answer must of necessity be an individualism.

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[9] *N. Y. State Hosp. Bull.*, ii, p. 269, 1914.

We will postulate as the normal individual one who is possessed of a properly balanced organism including within the scope of such a conception not only the brain but all the organs of the body. This should be our viewpoint toward that very much abused term personality. No personality can be considered at all if attention is paid only to the mental reactions of the individual. It must be stated that the mental reactions of an individual depend in a great part on the state of his reactions at physiological, chemical and physical levels. A man's liver is as much a part of his personality as his memory or his emotional balance. With this in mind, we will describe, therefore, as the normal, that individual whose organs react physiologically to internal and environmental stimuli. Such individuals constitute a large part of the population and tend to gather in the largest groups. The personality of a group of individuals brought up in one locality will differ from that of another group brought up in an entirely different atmosphere. The East-side boy eating East-side food, meeting people of average or less than average intelligence, honesty, and morality, will possess a very different personality from the boy brought up in another part of the city, surrounded by individuals who are living intellectually, physically, and physiologically at a much different level. Nevertheless, these two hypothetical individuals would have to be considered normal personalities and it is ridiculous to study individuals in any other way than this. In order to understand the patient who comes to see us it is essential that a great deal of data be obtained regarding, not only the development of the patient's personality but also regarding the environment in which that personality was synthesized. Wide variations are possible within normal limits. Without a great deal more information than we usually can get from clinic patients, it is impossible to determine whether a given individual is on the borderline of the normal or is about to become a psychotic. It is a question whether the mythical borderline psychoses really exist and whether what we have to deal with as such, are really not cases of variations in personality development and not actual psychoses. Such variations may be determined by maladjustments and have their origin in defects at the instinctive level or the maladaptations may be seen to exist at a physiological or physical level. In discussing instinctive reactions, the narrow view which makes a religion or a philosophy of life out of one of them and refuses absolutely to yield an equally important place to the rest is much to be condemned. A number of instincts, aside from that of sex, necessarily play an important part in determining individual reactions. With this introduction we will proceed to give by clinical presentation examples of two cases, one of maladaptations of psychogenic origin in which a number of instincts are defectively controlled, and another which seems to have a definite defect at a physiological and physical level. As an example of the first I wish to present the case of E. K. because it illustrates a type of reaction which is quite often seen in individuals possessing the sort of make-up pictured here. We frequently see jealous, fault-finding wives or husbands who are the cause of constant marital squalls. Careful study of this case will enable us to arrive at a plan whereby we can help to adjust these individuals. As this case is typical, we present it in full. We do not feel that it represents a definite disease complex but merely a paranoid-like reaction in an individual with a pathological personality.

CASE I

The patient is forty-eight years of age, female, married. She is the mother of eight children, five of whom are living. Three of these five were born deaf and dumb. She suffered from pneumonia fifteen years ago but since then has been well up to four months before her visit to the clinic. She had her menopause ten years ago.

For the last two and a half years she and her husband have been caretakers of a doctor's office, where a dentist also lived. Shortly after beginning her work here she says she heard the doctor and the dentist talking together about having intercourse with her. This they did any time they met and kept it up for over two years. About this time she developed severe headaches which are still present. Three months ago she told the doctor that he and the dentist should stop talking about her as her head was hurting her. Since then they have not talked about her. Twenty years ago her husband says she had a similar attack in which she accused a woman, a neighbor, of conspiring with her husband to defame her and poison her. She has always been jealous of her husband, suspicious of strangers, fault-finding, seclusive, and has always blamed him or someone else for all her troubles.

She sleeps poorly, her appetite is poor and her bowels are constipated. She is always depressed and apprehensive, has great difficulty in meeting people and likes to be alone. Memory, orientation, judgment, and the emotions do not seem to be disturbed. The patient is an efficient, painstaking, careful housekeeper and the physician for whom she works assured me that she is a very valuable, steady and intelligent worker. There are no evidences that she is deteriorated in any way whatsoever, mentally.

HISTORY.—*Previous History*. She was born of Hebrew parents in a small village of Hungary. The father died of cholera when she was seven years old. The mother was poor and had to work hard on a small farm and the children had to work hard, early and late, with no opportunity for pleasure or education. She learned to read and write but can perform the former skilled act much better than the latter. She came to the United States when she was fourteen years old, accompanied by a sister who was two years younger. Her mother had come over one year before. During the year she was alone in Europe she was cared for by a married sister. She went to work as a servant when she was fifteen years of age with a Jewish family in this city. The patient says she has had very little fun all her life and that it has continued to be a hard struggle with very few joyful or restful spots in it. She had little opportunity to cultivate the few friends she had and enjoyed her mother's and sister's company more than she could say. She was always sad, cried easily, was extremely sensitive and liked to go off by herself when she had the "blues" which was often.

She worked with the Hebrew family for two months and then left because they did not pay enough. She did not like to be a servant; she felt the inferiority and wanted better remuneration, consequently she changed places frequently. She wanted to work with her mother and sister in the shop but could not do the needlework because her eyesight was poor. When she was seventeen years of age her mother died of pneumonia. The patient cried and felt very badly for some time and after this was even more depressed than usual for a few years.

A match was arranged and she, like a dutiful sister, married the man who was picked out for her by her sister and her husband's parents. She did not love him and married him, as is the custom, after seeing him only twice. She has never been able to get along well with him and according to her story has always had a hard time keeping the home together because he was lazy and indifferent and a poor provider in many ways. They have had to move every three or four months because he could not get enough money together to pay the rent. Sometimes she would have to go out working to help pay the rent and to make ends meet. He was weak sexually. Physically he always suffered from sore throat and was not strong. The first child was born deaf and dumb and was followed by three others, each blow being harder to bear than the last. She was terribly shocked by these occurrences and blamed her husband because she reasoned that she and her family were always healthy while her husband had always been a semi-invalid and had suffered continuously from sore throat. She thinks that his sickness was the cause of her children being born deaf and dumb. The last two children were born normal but she is not able to explain this except by saying that two healthy children might accidentally come out of a total of eight—six, including a pair of twins, being born deaf and dumb.

She describes the doctor for whom she works as caretaker as a "nice young man but fond of fooling" and she is "too old and does not like fooling." When she got sick she would not let him examine her because he was "too fresh." Pressed for a more definite explanation she accuses him of boasting to his patients that he could get the best of her. About a year ago she noticed that he would smile and laugh boisterously at her and then heard him go into the consulting room and tell his patients, who were often sailors from the battleships nearby, of the old lady upstairs who was "stuck" on him and with whom he was going to "do business" some day. On other occasions she heard the doctor converse along the same lines with the dentist who rooms with him and who also joined in defamatory remarks, laughing and cracking jokes about her. Finally the doctor's remarks and smiles and laughing became unbearable to the patient and she spoke to him about it. She says that since then although she knows he still says these things about her, she no longer hears him. The patient also dislikes the doctor's wife who is young and pretty because she is too haughty and is overbearing. She says the doctor is foolish because he tells his wife everything and often as she opens the door for the patients to come in, and passes by their door she will hear the pair giggling and laughing about her and they will joke about the silly old fool upstairs who is in love with the doctor. When she was sick the wife wanted her put out, and once she heard the doctor tell the wife that "he could have the patient any time he wanted her." One day when the patient was very sick the doctor, instead of listening to her chest and examining her stomach, which she complained of, immediately made unnecessary advances which she took as attempts at suggesting coitus but she repulsed him saying that while "he was smart and young he could not have her."

Sexual History.—She began to masturbate when she was twelve years old; would go somewhere so that she could be alone to do it, and worried and was depressed afterwards because of religious compunctions and scruples. She was always "passionate" and never felt that her husband satisfied her desires. (Information voluntarily given without leading questions on third visit.) She continued to masturbate until one year ago. Her husband has not had intercourse with her for several years, she says. Her husband thinks that she has been unreasonable in this respect and has made impossible demands on his sexual strength. He is physically not well.

Physical Examination.—Examination discloses absolutely nothing. The heart and lungs are normal and the other viscera show no evidences of disease or injury. There are no palpable glands and the blood pressure is 145 systolic and 90 diastolic. The examination of the fundi reveals nothing, according to Dr. Holden, except that there is evidence of a marked hyperopia and presbyopia. The examination of the urine and blood Wassermann is negative.

SUMMARY AND ANALYSIS OF SYMPTOMS

A consideration of this case immediately shows that according to her husband she has been demonstrating paranoic reactions and has had delusional trends of a suspicious and jealous type referred to her husband and other people for years. She is probably a case of late paraphrenia. She still has all of her higher mental faculties practically undulled. She represents, too, a type of individual that is seen quite frequently. The jealous, fault-finding suspicious wife is not unusual and situations such as arose in this case have wrecked many homes. The synthesis of her defective personality is undoubtedly to be found in a study of the history of her childhood. Her parents were extremely poor, the father, the provider, died when she was very young. She and her sister and her mother had to struggle early and late on their small farm to make things go, and there was little time for social, educational, or mental development. The intellectual and affective responses were necessarily limited to such as might find outlet in contact with the mother and sister. She played but seldom, not only because there were no children to play with but she was usually so tired and overworked that play did not seem attractive. Soon she became introduced to her instinctive sexual life. Unrecognized and little understood, sexual cravings and impulses were finally satisfied by active, prolonged and continued masturbation. It was impossible for her to talk these matters over with her mother and sister, the religious and cultural compunctions were too great. She soon became over-developed in her sexual reactions and her intellectual processes revolved about her masturbatory activities, that is, her conscious thought processes were naturally limited and she grew to be a quiet, sullen, irritable, nervous woman, the prototype of a great many that we see in our clinics daily. Forced to marry a man chosen for her by her sister, because of faithful observation of custom, she soon began to get into difficulties. Her husband was physically weak, her sexual desires were strong; he was not able to satisfy them. He did not provide well for her, thus producing a defect of adjustment along the instinctive levels of nutrition and self-preservation. There was a constant struggle to live and to make both ends meet. She became moody and depressed and as she herself says, she has really never been happy at any time in her life. As an added factor came the birth of the deaf and dumb children, and then occurred the first opportunity for displacement of her affects. She blamed her husband for everything; he was responsible for her unhappiness; there must be something wrong with him or the children would not be born defective. He had deceived her as to his physical condition. He was weak and sickly and should never have married her; she could not love him, therefore, and possibly the doubt came into her mind that it was not only because her husband was physically weak; it was barely possible that instead of giving her all his sexual strength, he was distributing some of it to others more favored. Therefore, she accused him of improper relations with a life-long friend and benefactress known to both of them. This first episode occurred ten years ago. On several occasions since then this reaction was repeated with other women. Eventually, her present mental defect was precipitated by continued daily contact with a good-looking young physician who stood in a dominant relationship to her. He was her employer; he was energetic, possessed a pleasant personality and was always courteous and sympathetic towards her. She has volunteered frankly that love matters have always interested her. The ordinary, gentlemanly interest that the doctor showed in the patient was misinterpreted by her only too willingly. She thought that his pleasant nothings were improper insinuations. The next step was a suspicion as to his intentions; it was unbearable to her sense of personality to admit that it was *she* that desired his love, so she suspected *him* of dishonorable intentions. She worried a great deal about these things and spent many sleepless nights. She spoke about this matter to her husband, but he made little of the idea and laughed at her. She then had her hallucinations and delusions in which the doctor took the leading part.

The therapeutic result in this case is interesting. No analysis was attempted. The case was handled as follows: I had a personal conversation with the physician with whom she lived and advised a plan of action. I cautioned him against his too social and too generous treatment of the patient. I advised that he be firm but fair with her; it was thought best that he insist on her taking no authority beyond what was prescribed for her. No conversation or discussions were to be had between them excepting on matters of business. If she was around within hearing distance and it was necessary for him to laugh or make pleasant remarks to a patient or to his associate I considered it essential that such remarks be made in a matter of fact way or in such a manner that not even her suspicious nature could take them as referring to her. I counseled that everything be done in an open, frank manner, that all relationships between them be frankly discussed. I counseled also that the doctor's wife might help by not poking fun at her for being foolishly in love with him and that she should act in a thoroughly dignified manner in all her relations with the patient. The husband was cautioned not to ridicule the patient, but to be conciliatory and to explain apparently suspicious happenings. After several months of this plan of treatment, which was reinforced by no other measures of which I know, this patient recovered from her attack and has either submerged her delusions or has adjusted herself and is now in a condition which, for her, is normal.

CASE II

The next case which illustrates a defect at the physiological level has to do with a young married woman, aged nineteen, who came to the clinic complaining of "nervousness." She said that she had had during the last year a complete change of character. She had been married a year and a half before and had considered herself very lucky in obtaining a sober, hard-working mechanic as a husband, who not only was kind to her but also provided fairly well the necessities of life and even some luxuries. Having been jolly, happy, frank and full of the joy of life, she had gradually become nervous, fearful, irritable, sensitive, and complained of a series of symptoms. Her face was flushed, her hands were cold, and she perspired easily. She suffered from a great deal of leucorrhea. She was constipated, passed water frequently and had attacks of indigestion with much gas. She also felt a palpitation of the heart and complained of definite changes in her menstrual history. Her menstruation before the onset of her illness had been regular; the interval being four weeks, duration eight days, quantity moderate; now the interval was two or three weeks, the duration three days, and the quantity scanty. She was afraid to be alone; she feared that she was going insane. At times, particularly when she was alone, she felt as if she must scream or die. During the past week she had vomited three times and had had headaches, both symptoms occurring in the morning. Her breasts had become enlarged and felt hot. She has recently lost her appetite.

The physical examination was negative. No gynecological examination is permitted in our clinic, but the suspicion was nevertheless justified that the patient was pregnant. Whether she is pregnant or not makes little difference in this discussion. The blood pressure was 110 systolic and 70 diastolic; the pulse was 90 and there was a slight prominence in the eyeballs. No Mœbius or von Graafe sign was present, and there was no evidence of enlargement of the thyroid gland on palpation or inspection. A careful neurological examination showed nothing except hyperactive and equal reflexes.

An important fact is that the symptomatology presented by this individual shows marked evidences of a physiological change. Whether it be due to an early pregnancy or to a disturbance of the glands of internal secretion makes no difference. The gynecological department reports that there is no obstetric or gynecological condition present (February 13, 1918). The fact nevertheless remains that the reactions which this patient presents, and which can be labeled either anxiety neurosis or hyperthyroidism, are undoubtedly influenced if not entirely caused, by the physiological changes which are taking place either as the result of the pregnancy or as a result of disturbances in ovarian and corpus luteum internal secretion. A great many of her symptoms may be explained on the theory of sympathetico-tonia and vagotonia. We know that in a large sense the sex glands are not limited to the ovaries in women and testicles in men, but that the adrenals, the pituitary, and the thyroid glands play an important part, not only in the sex function, but in the development of secondary sex characteristics. The only reason why sex is mentioned at all in connection with this patient is the fact that she has of late cultivated an actual repugnance against coitus; she voluntarily complained of this and it is not necessary to begin to delve into her "subconscious" in order to find mechanisms to explain it. It is only necessary to call attention to the hypopituitary types of asexuality, not only so far as body configuration is concerned but also in reference to the absence of sexual desire, to suspect what has happened here.

This case is not reported *in extenso* with a complete anamnesis and investigation of the development of the personality because there is every reason to believe that this patient, so far as her psychic characteristics are concerned, particularly her ability to adapt herself mentally, was undoubtedly normal before her marriage. She is confirmed in her statements that she was an average, contented, happy, healthy, evenly balanced girl before marriage. The change in her which puts her so much out of touch with her environment is brought about by her physiological condition and must be considered in the light of an essentially physiological reaction. This case could be much injured, by an extensive analysis, according to the principles of Freud and others. The husband should be instructed to live apart from the wife so far as sexual contact is concerned, for several months. Proper dietetic and hygienic measures should be instituted to control the vomiting; rest, and mild sedatives would seem to be indicated to control the palpitation, rapid pulse, and restlessness. There is no doubt that she will make a physiological adjustment.

In closing, it must be remembered that the abnormal mental reactions presented to us as borderline psychoses must be considered from every possible angle. Investigation should never be entirely psychobiological, psychological, physiological, or physical but rather a combination of all. Advances in medical science are often retarded by narrow and restricted points of view. We all would do well to study our clinical cases of mental maladjustment in the light suggested. Too many times we have seen mental cases attending our clinics with the physical examination chart almost entirely neglected. Such investigation of cases is prone to lead to error and is no doubt responsible for the many psychological sects, religions, and philosophies which are constantly being put forth as the truth in relation to the pathogenesis of the neuroses and functional psychoses.

A NEW CLINICAL TEST FOR TEMPERATURE SENSITIVITY

BASED ON THE REACTION TO COLD AND HOT STIMULI SIMULTANEOUSLY APPLIED

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GENERAL OUTLINE

- Historical
- Experimental
  - Statement of problem
  - Apparatus
  - Material and Method
    - Subjects, Areas tested, Arrangement of Series, Temperatures used
  - Procedure
  - Conditions affecting results
    - Limitations of method
    - Changes in temperature of skin or stimulus
    - Topography of warm and cold spots
  - Results
    - General character of reactions to double stimuli
    - Proportion of responses giving *cold* first
    - Proportion of responses giving only one sensation
  - Reaction to single stimuli
    - Overlapping of reaction-time to cold and hot
    - Responses giving both sensations
  - Detailed consideration of responses to double stimuli
    - Responses giving *hot* first—comparison with those giving *cold* first
    - Responses giving *cold* first
      - General distribution
      - Effect of double stimulus on reaction-time to hot
  - Summary of individual cases
- Conclusions

HISTORICAL

A review of the literature on the temperature senses is a task of rather large dimensions. There are so many factors, however, influencing any investigation in this field, and there is so little in the literature that bears specifically on the present problem, that I shall attempt to give a somewhat sketchy survey of the whole field.

The problems attacked have been many, the methods of approach various, and the conclusions of all possible degrees of diversity; so that it is not always easy to give a very clear presentation. Indeed, one fervently reëchoes the words of Trotter and Davies: "The study of thermal sensitivity is a peculiarly difficult one, not only in regard to technical detail, but especially in regard to obtaining clear conceptions and expounding them without confusion."[1]

There seem to be four general classes of problems that have been investigated in regard to the temperature senses—four general methods of approach, which while not mutually exclusive, are sufficiently distinct to give a certain coherence to the discussion. These four classes are:

1. *The psychophysical*—under which I have classed the various problems of *sensitivity* that occupied so large a part of the earlier literature—problems of the threshold values of sensation, of discrimination, and of the limiting values where sensations of temperature pass over into those of pain; and the question of topography—the relative sensitivity of various parts of the body.

2. *The physiological*—which took its impetus from the question of the specific energies of the nerves, because of the apparent contradiction that cutaneous sensations offered to this theory. Under this class will fall the

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[1] *J. f. Psychol. u. Neurol.*, 20, 1913, p. E 106.

question of the nature and distribution of the end-organs (closely allied to the topography), the question of the nature of the arousal of the sensation and the consequent explanations of adaptation, and the question of the constitution of the nerves which was raised by the recent work of Head.

3. *The psychological*—which is, to be sure, bound up with all the rest, but under which I have chosen to consider the questions related chiefly to qualities of sensation and reaction-time.

4. *The clinical*—which is, to a great extent, merely the application of results obtained by other methods to abnormal cases. It has, however, set many of the problems under the other headings, and has, besides, its peculiar problems of sensory dissociation to treat.

PSYCHOPHYSICAL INVESTIGATIONS

It is quite fitting to begin from the psychophysical point of view, since the very first recorded observations are those of Weber (24), that a metal cylinder of 15° C. felt colder on some parts of the body than on others, and similarly, one of 40° felt warmer in some places than in others. Fechner, also, did not omit to test temperature sensitivity to corroborate Weber's law; but these experiments we may pass over, to consider the more specific attempts to measure temperature sensitivity.

Threshold values may here, as in other cases, be of three sorts, although our authors have not always distinguished them very clearly. The stimulus limen (R. L.) would correspond to the least difference from the skin temperature that could be sensed; the difference limen (D. L.) would correspond to the least noticeable difference between two stimuli at other than skin temperature; the terminal limen (T. R.) would be the limiting value where sensations of temperature ceased to be felt as such. The first experiment of this sort was that of Nothnagel (16), in 1866. He used two small cylindrical vessels filled with water, each fitted with a thermometer dipping into the water. The water was heated or cooled to the desired temperature by inserting into it a large key previously heated or cooled. The two vessels were applied successively to the same spot, and the discriminative interval determined. He made out discrimination tables for various parts of the skin surface, as a starting point for the investigation of pathological conditions, in which he was primarily interested. Discrimination, which he found to be best between 27° and 33° C., varied from .2° on the forearm, upper arm, and cheek—the most sensitive parts—to 1.2° in the middle of the back. At that time, of course, the separation of thermal sensations into two separate series had not been thought of; consequently, Nothnagel's values may be sometimes true difference thresholds and sometimes may represent the absolute threshold, or least noticeable sensation for cold or warm. After the distinction had been made, Eulenberg made an attempt to determine the least differences above and below the skin temperature that could be perceived as warm and cold respectively (7). Eulenberg's apparatus was somewhat more refined. He used two thermometers with flattened bases, one of which had its base wound with a platinum wire through which a current could be sent to raise the temperature of the base. The heat was controlled by a rheostat. One thermometer was placed on the skin and left until it registered the skin temperature; the other was then heated by the current to a point slightly above or below this and set down in the same place. Minimal differences perceived as cold and warm respectively were thus determined for various areas. The difference

R. L. for Cold and Warm (Eulenberg)

	Minimal Cold Difference	*Minimal Warm Difference*
Sternum	.2°	.2°
Fingers—dorsal	.2— .3	.2
Ball of thumb	.3— .4	.4
Back of hand	.3— .4	.4
Forearm—dorsal	.3— .4	.2
Forearm—volar	.4	.3— .4
Cheek	.4	.4
Upper arm	.5	.5
Top of foot	.5	.5— .6
Leg—upper part	.5— .6	.5— .6
Leg—middle part	.7	.7
Thigh—ventral	.6	.5— .6
Back—lateral	.9	.9—1.0
Back—median	1.0	1.1

for each sense was found to be practically identical for the same area of the skin, and to vary, for different areas, from .2° C. in the most sensitive parts (sternum, back of fingers) to 1° or 1.1° on the back.

The third sort of threshold—the pain threshold for thermic stimuli—was first investigated by Donath, whose work was published in 1884 (6). He contrived two instruments, one for investigating cold-pain, which he designated kryalgimeter (χρύος—cold, ἄλγος—pain), the other for warm-pain, which he called thermalgimeter. The kryalgimeter was a thermometer ending in a spiral mercury vessel which fitted exactly into an opening in a piece of heavy flannel. This was placed on the skin and cooled by spraying it with ether —the flannel protecting the surrounding skin —until a sensation of pain was felt. The thermalgimeter was a similar thermometer, ending in an elongated bulb which was wound with a platinum wire through which a current could be sent. This was placed on the skin, and the temperature increased until pain was reported. He found noteworthy individual differences among his six normal subjects and wide variations, according to the part of the skin stimulated, from —11.4° to 2.8° C. for the appearance of cold-pain, and from 36.3° to 52.6° for warm-pain. In general, ventral surfaces were more sensitive than dorsal, upper extremities than lower, left side than right; the ends of the fingers were the least sensitive. He concluded that the pain aroused by extreme degrees of temperature is a much grosser sensation than that from the middle intensities, and lacks the fineness of discrimination of the latter. The values that Donath obtained for cold-pain seem extraordinarily low, and suggest that his method may have overlooked the effect of adaptation: he does not state how long the contact with the skin continued. He does, however, say that cold-pain is much more difficult to recognize and define, and arises more slowly than warm.

Neumann, in 1903, investigated the same question, in regard to the warm sense only, in order to compare the interval between the sensation of warmth and that of pain (15). He used a metal cylinder partly filled with water that could be heated by an electric current. A thermometer was inserted into the water and attached to the handle; the handle also had a little plunger by which the water was kept constantly circulating, in order to assure an even temperature throughout. Neumann found that while the threshold for warm might vary as much as two degrees C. for the same individual in the same place at different times, the threshold for pain was fairly constant for the same place and varied according to the area stimulated from 35° to 52°—values practically identical with Donath's. Both Donath and Neumann were interested chiefly in pathological conditions, and their work will be referred to later.

The other question pertaining to sensitivity is that of the relative sensitivity of different parts of the body. Goldscheider was the first to make a systematic attempt to experiment upon this question. His earliest experiments were published in 1887 (9*a*). He explored the skin with a small metal surface that had been heated or cooled, and divided sensitivity into twelve degrees of intensity for cold stimulation and into eight for warm. It is clear, of course, that the actual temperature of the skin in this case is of great importance, and also the proportion of warm and cold spots in the various areas. Alrutz repeated topographical experiments after adapting each area tested to a temperature of 32° (2*a*). He was able to differentiate only three degrees of sensitivity; but he used a stimulus temperature of 34.5°, in order to have it below the point at which the cold nerves are paradoxically stimulated by high temperatures, while Goldscheider had used a temperature of 45°—49°.[2] Such radically different modes of procedure could not, of course, arrive at the same results. The truth probably is that these differences of sensitivity are not peculiarly areal differences, but are caused by differences in skin temperature and by the presence of more or less strongly reacting spots in certain areas. Whether these spots have any constant topographical distribution is doubtful; in any case, it could only be determined by a systematic exploration, point by point.

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[2] The difference is more than one of method. It is due to the divergence in the views of Goldscheider and Alrutz as to the nature of the sensation of heat—the former maintaining it to be just an intensified warm, the latter a fusion of warm and paradoxical cold.

PHYSIOLOGICAL INVESTIGATIONS

It was originally supposed that the same nerves conducted all cutaneous impressions. It was not until 1883 that Blix, who was interested to determine how well-founded was the contradiction that cutaneous sensations offered to the theory of the specific energies of the nerves, discovered that sensations from various kinds of cutaneous stimuli were limited to permanent spots—among the sensations, those of cold and heat (3). The same discovery was made independently at about the same time by Goldscheider (9*b*), and by Donaldson (5). Blix used an induced current with a pin for the stimulating electrode, and found that distinctly localized spots responded by sensations of pain, pressure, cold, and heat, and that these spots were never superimposed. Other methods of stimulation confirmed the results, and it was further observed that a cold spot never responded to a warm stimulus, nor a warm spot to a cold stimulus. Blix concluded that there were separate cutaneous nerves with separate terminations in the skin. He mapped out various regions of his own body for temperature spots and found them arranged irregularly in groups, distributed fairly uniformly over the body, most closely on the dorsal side of the hand and fingers, most scattered on the sacral region of the back, with cold spots always more numerous than warm. Donaldson's observations confirmed Blix's. Goldscheider found a much more numerous count, but his method was probably at fault, since later work has corroborated Blix. The latest and most complete observations were those of Sommer (19), reported in 1900. He found (for the benefit of anybody who is statistically minded) a total of half a million cold spots and thirty thousand warm—an average of 12–13 cold spots and 1–2 warm spots per square centimeter.

Donaldson made the earliest attempt to discover the end-organs associated with the sensations of warm and cold. He made a histological examination of a piece of skin that had previously been explored and marked and then excised, but his findings were entirely negative. He evidently did not cut deep enough, for he found that the recovering area was perfectly sensitive. Von Frey, whose work was published about ten years later, has done the most constructive work in the discovery of the nerve-endings (23). It is now generally supposed that the end-bulbs of Kraus mediate cold sensations, and the Ruffini corpuscles those of warmth.

It is generally assumed that the warm end-organs lie deeper than the cold, because of the greater difficulty in localizing warm sensations and because of their longer reaction-time. Thunberg pointed out that these assumptions were not sufficient proof; both conditions might be due to the nature of the end-organ—Goldscheider indeed had assumed that the warm end-organs had a longer latent time. Therefore Thunberg undertook an investigation of the question by an indirect method (20*b*). He applied silver plates of the same area but of varying degrees of thickness to the skin. The principle, if I understand it correctly, was as follows: Two plates of the same area and temperature, but of different thickness, will add warmth to, or withdraw it from the skin in different amounts, in a measurable proportion to the thickness. Since the skin-temperature is a balance between the temperature of the blood and the outer temperature in contact with the skin, the amount of warmth added to—or withdrawn from—the skin until the different layers of the skin reach a new temporary balance will be a measure of the depth at which the balance is attained. Consequently, the application of a very intensive stimulus of less thickness will have a greater effect on the more superficial layers than on the deeper-lying layers of the skin; while on the other hand, a stimulus of less intensity but greater thickness will give a relatively favorable opportunity for the stimulation of the deeper layers. Since then, as Thunberg found, the more intense, thinner plate provided the optimum conditions for the arousal of paradoxical cold sensations, while a stimulus of less intensity but of greater mass offered the best conditions for obtaining a strong warm sensation, he concluded that the cold nerve-endings lie more superficially, the warm nerve-endings deeper.

The question of the depth at which the end-organs lie is closely connected with the question, somewhat theoretical but nevertheless important in its practical bearing, of the nature of the arousal of thermal sensations. Does the end-organ change its temperature or does it remain constant? If it changes, do the changes take place in the same direction as the changes in the outer skin-temperature or in the opposite direction? It is generally considered that, since the skin-temperature is found to be differently tempered for changing conditions of temperature without arousing any sensation, there is a zone of indifference —usually called the "physiological zero"— which is capable of shifting up and down the scale within rather wide limits. But Thunberg points out (*20c*) that we have no proof that the temperature of the end-organs is actually the same that we obtain when we measure skin-temperature, or that it necessarily changes in the same direction. What we get by measuring the skin-temperature is a balance between the different layers of the skin, which are normally cooled from without by the air and warmed from within by the blood. But when the outer layers are cooled by contact with the cold, the deeper layers, according to Thunberg, have their temperature raised by an increased flow of blood to restore the balance; so a fall of temperature toward the surface takes place. Somewhere between, there may be a layer that maintains its own temperature. Since it is not known in what layer the end-organs are, it cannot be said what is the nature of the change. There are two views current: that of Weber, that temperature sensation is due to change of skin-temperature (or, as amended by Thunberg, to change in the layer of the end-organ) and lasts as long as the change lasts; and that of Hering, that temperature sensation is due to the actual temperature of the end-organ and lasts until the physiological zero reaches a new location—the temporary temperature of the end-organ. According to Hering, the shifting physiological zero—the adaptation to any actual temperature of the end-organ—is what accounts for the absence of sensation, though just what the physiological zero is is not clear. According to Weber, any relatively stable temperature following a period of change is the cause of the absence of sensation. The difference in the two views is due partly to differences in their findings as to the duration of sensation.

An experiment to determine the duration of a sensation with a constant stimulus was made in 1903 by Holm (10). He found that the duration of the sensation decreased as the temperature of the stimulus approached that of the skin, and he thought this suggested that the duration of the sensation probably agreed with that of the temperature change, since prolongation of the stimulus did not prolong the sensation. He found no after-images with stimuli of middle intensities, but extreme cold gave rise to an after-image which appeared some moments after the removal of the stimulus—due, Holm thought, to paradoxical cold sensations occasioned by a quick warming of the cooled cold-organs by the blood. The absence of after-images he thinks is a further support of Weber's theory; because if the absolute temperature were effective, one would expect after-images with weak as well as with strong sensations.

DURATION OF SENSATION (Holm)

Cold

Temperature	*Average Duration*
30°	31 sec.
25	47
20	72
15	112
10	165
5	210

Warm

Temperature	*Average Duration*
40°	126 sec.
45	152

There is a recent study on the effects of adaptation by Edwina Abbot, which suggests a somewhat different conclusion (1). She made four groups of experiments for four conditions of adaptation. In the first group her subjects had their hands adapted to a temperature of 40°, by immersion in warm water. Then the D. L. was determined by dipping the two hands into separate jars, for 37.5°, 35°, 32.5°, 30°, 27.5°, 25°, 22.5°, 17.5°. She found a "consistent drop" in the D. L. at 32.5°. In the second group, she determined the D. L. for 40°, 32.5°, 27.5°, 25°, after adaptation to various temperatures. She found that the limen for 32.5° varied hardly at all under the varying conditions of adaptation. This seemed to point to 32.5° as a condition of adaptation suitable for a norm by which to compare the limena for different temperatures under other conditions of adaptation. In the third group, to test this, she compared the D. L.'s for various temperatures after adaptation to 40°, 32.5°, 17.5°, and found that the D. L.'s for 32.5° under the three conditions of adaptation were within .5° of each other, while for other temperatures the D. L. rose after adaptation to 40°, and fell after adaptation to 17.5°. The fourth group was undertaken to test the possibility of a double adaptation—an adaptation to both extremes at once. In this group, she compared the effect on the D. L.'s for various temperatures of adaptation to 40°, with the effect of a double adaptation, to 40° immediately followed by adaptation to 17.5°. The values for the D. L. were practically alike in this experiment for extreme temperatures, but between 35° and 27.5° the D. L. was higher after double adaptation, except again, at 32.5°, where the value was about the same for both conditions. This was considered to confirm the hypothesis. All of Dr. Abbot's results suggested that varying conditions of adaptation had practically no effect on sensitivity at normal skin-temperature, and she concludes that "persistence of skin-temperature as a more or less constant basis for appreciating variation of temperature is a much more possible conception than the . . . physiological zero," "since it does not fit the facts . . . and since the facts it attempted to explain may be explained without it." Dr. Abbot's observations were not very numerous, however, and her results are suggestive rather than conclusive.

There is one other question to be considered under the physiological caption—the question of what constitutes an adequate stimulus for warm and cold spots. Blix had first used an electrical stimulus, and found both warm and cold spots sensitive. Kiesow (11) found temperature spots likewise sensitive to electrical stimulation, and to mechanical stimulation—if the observations were carefully made, if the observer was sufficiently practised, and if conditions were otherwise favorable. That is, he obtained responses to mechanical stimulation in nine out of thirty tested cold spots, in twenty-one out of forty-six trials; in ten out of thirty trials with warm spots for one observer, five out of fifteen for another, and seven out of ten for a third. The percentage of successes is not startling, but is enough, Kiesow thinks, to prove mechanical stimulation possible. Goldscheider (9*c*), Alrutz (2*c*) and others have confirmed the possibility of mechanical stimulation, though Head (18) finds no evidence of it. The earlier investigators had found that cold spots never responded to warm stimuli, nor warm spots to cold. Kiesow tested the spots by means of the "opposite stimulus" and got a rather curious result. He never got a cold sensation by cold stimulation of a warm spot, but, on the other hand, he found scarcely a cold spot that would not respond to temperatures of 47° to 50° by a sensation of warmth. This, Titchener says, is a misreading of the facts: "In reality, radiation occurred, and the process observed was not that of warmth but of heat."[3] However, paradoxical sensations of cold—*i. e.*, cold sensations from a warm stimulus applied to a cold spot—were discovered by von Frey (23), and confirmed by Thunberg (20*a*), Alrutz (2*d*), and are admitted by all. Paradoxical warm sensations have been described by Goldscheider, but no other investigator has been able to discover them.

PSYCHOLOGICAL INVESTIGATIONS

It is perhaps somewhat arbitrary to attempt to separate the psychological questions from the physiological, but I have reserved this designation for the investigations relating chiefly to the question of the nature and quality of the sensation of heat—the question that has loomed largest on the psychological horizon and that brings about a triangular debate between Thunberg, Alrutz, and Goldscheider—and for the determination of the reaction-time to cold, warm, and hot stimuli.

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[3] Titchener, 'Quantitative Instructor's Manual,' p. 86.

Paradoxical cold sensations are the crux of this discussion. While everybody admits their existence, there is considerable diversity of opinion as to the exact manner of their arousal and the interpretation of their significance. Goldscheider, who originally believed they were due to mechanical stimulation, now admits that they are truly paradoxical, but difficult to arouse except from very sensitive cold spots (9*d*). He says they never appear with a stimulus of less than 38°, and are not a frequent phenomenon with temperatures below 41°–45°. Alrutz and Thunberg, however, find paradoxical cold sensations a constant phenomenon from temperatures of 36°–38° and up (2*b*, 20*a*). Goldscheider also reports paradoxical warm sensations from cold stimuli (of 21°–27.5°); consequently, the significance of paradoxical sensations for Goldscheider is simply as another means of confirming the specific nature of the end-organs. In addition to paradoxical cold sensations by point stimulation, Alrutz and Thunberg showed that they could be obtained also by areal stimulation with an object at a fairly high temperature, if the area was first adapted to moderate warmth for two minutes. These facts, and the absence of paradoxical warm sensations, in their observations, led these two investigators to the belief that paradoxical cold sensations were an essential element in the sensation of heat. Alrutz found two facts in his earlier work that confirmed this: first, that on areas of the skin where cold spots are lacking, stimulation by any degree of heat produces warm sensations and pain sensations, but never heat; second, that on areas where cold sensitivity was strongly developed but warm sensitivity weak, sensations of heat could be experienced but never strong warm sensations. In a later paper (2*a*), Alrutz added a third proof that the hot sensation involved the stimulation of cold nerves, from experiments in reaction-time of warm, hot, and paradoxical cold sensations. Alrutz found that hot sensations had a characteristic quality, which he called *Anschwellung*—a sort of crescendo effect from the first perception of warm to the full perception of hot. This meant that the reaction to warm was quicker than that to hot. The average reaction-time for warm at a temperature of 42.5°, is 385σ; for hot at about the same temperature, 795σ; for warm at 43–44.5°, 267σ; for hot at 43–46.5°, 624σ. This longer time for the perception of hot Alrutz finds to be parallel with the reaction for paradoxical cold sensation—745σ for a stimulus of 42°, 750σ for a stimulus of 51°.

Alrutz's figures are based upon rather few observations, but he compares them with those that Goldscheider had previously obtained, and finds them quite similar. Goldscheider had found a quality in the sensations he calls warm, similar to the *Anschwellung* of Alrutz's hot sensations—that is, a gradual rise from the "minimum" to the "maximum" of intensity.

REACTION-TIME TO HOT (Goldscheider, Alrutz)

Goldscheider

	Average	
Temperature	*Minimum*	*Maximum*
35°	750σ	
39	371	$1{,}105\sigma$
40	345	777
44	282	585
49	283	456

Alrutz

Temperature	*Average Time*
36.5°	805σ
38.75	710
43.5	576
46.5	517
50	544

Alrutz points out that Goldscheider's figures show, as he contended, a greater effect of the stimulus temperature upon the hot reactions than upon the warm—*i. e.*, a greater decrease in the reaction-time to the maximum than to the minimum, with increasing intensity of the stimulus. Since Alrutz found a parallel decrease in the reaction-time to paradoxical cold, as the stimulus temperature rose, he considers this conclusive evidence that the sensation of heat is due to the simultaneous stimulation of warm and cold nerves.

So far, Alrutz and Thunberg agree, but they differ as to the quality of the sensation. Alrutz considers it an unanalyzable fusion. His introspective reports show, he says, that hot is, qualitatively, a sensation having the same relation to warm and cold that orange bears to red and yellow, similar sometimes to one, sometimes to the other, but never separable into the two components. Thunberg, on the other hand, calls hot an analyzable fusion —he thinks it possible to separate the element of paradoxical cold from the dominating hot sensation. At the third corner of the triangle stands Goldscheider, who considers hot simply as an intensive warm sensation, quite independent of paradoxical cold. In the study published in 1912 he takes up the argument of Alrutz point by point, and attempts to demolish it. In regard to the first argument, that on areas where cold spots are lacking no hot can be felt, Goldscheider finds that practically no such areas exist. (It will be remembered that he found a more numerous count of temperature spots than other investigators.) And if a refutation of the argument is needed, Goldscheider finds that in regions where there are sensitive warm spots, with scattered, not very sensitive cold spots, he is able to get quite as vivid hot sensations as on areas provided with strongly sensitive cold spots. The second argument, that regions that possess strong cold but weak warm sense will give hot sensations but never strong warm, Goldscheider meets again by the opposite experience: he gets warm and burning sensations but never hot. As for the third argument, that reaction-time for paradoxical cold agrees with that for hot and that therefore paradoxical cold is significant for hot sensation, Goldscheider considers this observation doubtful because of its great difficulty (he points out that only Alrutz himself made these observations), and that even if true, it is no proof of identity. He thinks the phenomenon much more explicable on the theory that a certain intensity of effect of a warm stimulus—which naturally must depend on a certain duration of the contact—raises the intensity of excitation of the warm nerves to the state where they give a hot sensation and that this intensity also provides the condition by which cold nerves are stimulated by warm stimuli. Other considerations too, especially that the hot sensation is strongest on areas where warm sensitivity is most strongly developed, persuade Goldscheider that Alrutz's theory is untenable. To this, of course, Alrutz would reply that Goldscheider's determination of warm sensitivity was made by hot, not by warm stimuli, and that this is a circular argument. The question cannot be decided until unprejudiced observers have contributed enough evidence to outweigh these contradictory findings. At the present time, I think it might be said that psychologists incline to agree with Alrutz, while physiologists find themselves in accord with Goldscheider.[4]

The most systematic work in reaction-time to sensations of temperature has been done by Goldscheider (9*c*, p. 312 *et seq.*). He devised a rather ingenious device for measuring the time. The stimulus, a small brass sphere into which a thermometer was inserted, was arranged by means of levers so that its contact with the skin closed an electric circuit, its removal broke the circuit. The making and breaking of the current were noted by an electric marker on a smoked drum of a kymograph, on which a time marker, controlled by an electric tuning fork, indicated hundredths of a second. He found that the reaction-time depends upon the intensity of the stimulus, upon the threshold value of the sensation, and upon the sensitivity of the part of the body tested.

The difference in reaction-time of different parts of the body is shown in the following table:

	Stimulus 15° C.	*Stimulus 50° C.*
Face	.135 sec.	.190 sec.
Upper extremity	.150	.270
Abdomen	.226	.620
Lower extremity	.255	.790

The effect of the stimulus-temperature is shown in the following table. The region tested was the left forearm—an area moderately sensitive to cold, with a skin-temperature of 32.5°, and an area quite sensitive to warm, with skin-temperature 32.7°.

Stimulus-Temperature	*Cold Reaction*
30—29.5°	.399 sec.
28.5—28	.381
20—19	.377
15.5—15	.278

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[4] Except those who follow Head's theory of the constitution of the nerves; but Head, too, while he separates the mechanism for warm and hot impressions, postulates a simple sensory "heat spot." *Cf.* p. 128.

Stimulus-Temperature	*Warm Reaction*	
	Minimum	*Maximum*
35°	.750 sec.	
39	.371	1.105 sec.
40	.345	.777
44	.282	.585
49.5	.283	.456

It may be noted that for cold there is a marked drop in the reaction-time at 15°, while for warm the most marked drop occurs at 39°. The latter point is very much nearer the skin-temperature than the former—a result that, if constant, is a little puzzling.

CLINICAL INVESTIGATIONS

Following Fechner, interest was much stimulated to the investigation of thermal sensations. Nothnagel pointed out in 1866 that temperature sensibility was the first to be disturbed in diseases of the brain and spinal cord and in neuroses. He established norms of discrimination for various parts of the body, as has already been explained, with which clinical cases might be compared. He found hypo-esthesia to be the general rule—a raising of the threshold for discrimination. Often, however, in pathological cases, an apparent hyperesthesia occurs—an increased *effect* of a given temperature, apparently greater sensitivity—while discrimination remains normal or is reduced. This method did not prove very valuable, being too laborious and unnecessarily fine.

Donath next devised a method, on the assumption that the important thing clinically was to discover where temperature sensations passed over into pain. The method has been described. He applied it to the examination of thirteen cases of tabes, but curiously, he did not examine the same cases for both the warm-pain-threshold and the cold-pain-threshold. He found that warm-pain usually appeared at a higher temperature than normally, and cold-pain usually (though not always) at a lower. He concluded that the pain aroused by cold and warm was a much grosser sensation than the perception of moderate degrees of temperature and lacked the possibilities of discrimination of the latter. Neumann also investigated the pain threshold for warm stimuli. In four cases of tabes he found the thresholds for warm and for pain higher than normal; in four cases of spondylitis he found two hypoesthetic in the affected region; in six cases of hysteria he found three normal, two hypoesthetic, and one hyperesthetic.

Goldscheider thought that the sensitivity of different areas would be a good clinical test (9*a*). He made a topographical map of the body on the basis of the intensity of sensation from the same stimulus, and then, in cases of disease, compared parts that normally had a constant relation to each other, to see if the sensitivity was affected. Obviously, the lack of any control for the introspective comparisons of clinical subjects would make this method of slight value.

The next clinical attempt was Leegaard's, who took as his point of departure the fact of the indifference zone—the region of the scale at and near the skin-temperature which is not felt as either cold or warm (13). He used little copper cylinders which were applied to the same spot for five seconds each, beginning at a temperature of 20°–25° C., and ascending by steps of .5° until the lower and upper limits of the indifference zone were reached. He found that normally the indifference zone was not over 1°, but in cases of brain and cord lesions and peripheral affections that he investigated, it might vary to any extent. In one case of hemiplegia, for instance, he found an indifference zone of 26°, in a case of transverse myelitis, an indifference zone of 14° in the affected region. These are rather interesting, because, while Leegaard did not differentiate two senses, and while his procedure was somewhat questionable, his experiments in a way anticipate those of Head.

The possibility of using tests of cutaneous sensitivity as a means of differential diagnosis, in the case of lesions of the nervous centers, is partly effect and partly cause of the more recently increased knowledge of conduction paths. Laehr did some important early work in distinguishing different types of partial sensory dissociation (12). In particular he pointed out the so-called syringomyelic type of dissociation, which is characterized by a disturbance of pain and temperature and the preservation of contact and pressure sensations. By 1905, four distinct types of dissociation had been described. Marinesco (14) classifies them as follows:

(1) Deterioration of all forms of cutaneous sensibility.

(2) Abolition of pressure and vibratory senses (or one of these with conservation of all other forms).

(3) Abolition of thermic and pain senses with conservation of all others (syringomyelic type).

(4) Impairment of thermic, tactile, and pain senses with conservation of pressure and vibratory sensations.

An entirely new direction of work in the sphere of sensory dissociation has been suggested within the last decade by the work of Head and his colleagues. Head had the radial nerve in his arm divided, a small portion excised, and the ends sutured. Then he studied the return of sensation to the area of distribution of the nerve. His most complete report (18) was published in 1908. His findings were that sensitivity returned gradually: first, to pressure, cutaneous pain, and extremes of heat and cold, accompanied by the phenomena of intensification and peripheral reference of the sensation; and after a considerable interval (160 to 400 days) sensitivity to light touch, to moderate degrees of temperature, and the discrimination of two points simultaneously applied. His conclusions are familiar: that the skin is supplied by two systems of nerves—one providing for a more fundamental, reflex, diffused response to painful stimuli and the extremes of heat and cold ("protopathic sensibility"); the other producing localized responses, discrimination of light touches, of two points simultaneously applied, and of intermediate degrees of temperature ("epicritic sensibility"). More specifically, in regard to temperature he found that on the recovering area of the hand that was in a "protopathic" condition, punctate stimuli of about 17° C. were the first to be sensed; a little later, a stinging sensation in response to punctate stimulus of 50°, and then a sensation of heat from a stimulus of 45°. As sensitivity increased, the limits approached slightly nearer, but at no time while the hand was in a protopathic condition (*i. e.*, while it was insensitive to cotton wool and unable to discriminate two compass points), did any cold spot react to a temperature above 26° (except paradoxically to a hot stimulus), nor did any "heat spot" react to any temperature under 37°. Only particularly sensitive spots reacted to these temperatures: the usual limits were about 20° and 40°. But simultaneously with the restoration of sensitivity to cotton wool, an areal stimulation (by a surface of 1.25 cm. diameter) of 36° to 34° gave a warm, well-localized sensation from parts where no "heat spots" had been discovered. Still, however, punctate stimulation of the "heat spots" gave no sensation if the temperature was under 38°. With cold, the experience was not quite so clear but seemed to Head analogous. With the appearance of the finer discrimination, no increase in the number of heat spots was observed; on the contrary, the separate spots lost their increased vividness. The recovered sensitivity to moderate stimuli, Head thinks, cannot be due to the regeneration of the nerve, because he finds that a similar condition obtains on the normal skin. If he marked out all heat and cold spots in a normal area, and then applied a stimulus between 26° and 37° to a small area where no spots had been found, such area would nevertheless respond with the appropriate sensation. He concludes therefore, that there are two mechanisms, one punctate, which responds to temperatures below 26° and above 37°; the other not punctate, which responds to areal stimulation between these two points.

The facts of adaptation Head finds offer further proof of different mechanisms. That epicritic sensibility depends on the temperature to which the skin is adapted, while protopathic sensibility does not, he concluded from the following observation: If both hands were adapted to water at 50°, a copper block at 29° felt cold to the normal hand, but gave no sensation on the protopathic area; similarly, after adaptation to 0°, it felt warm to the normal hand, but indifferent to the protopathic area. This is interpreted as meaning that the protopathic condition does not undergo the shifting of the threshold. When both hands were adapted to a cold of 10°, a paradoxical condition arose where 22° produced no sensation on the normal hand but gave cold sensation on the affected area. This is another evidence, Head says, of the fact that the protopathic parts are incapable of adaptation, and so allow the cold spots to react to 22°.

This experiment has been repeated by several investigators. Franz (8) supported Head's contention that different sets of nerves provide for the reception of extreme and medium stimuli, but he regards the difference in sensation as due to an overlapping of the nerve supply—a possibility that Head rejects. Trotter and Davies repeated the experiment under elaborate and stringent experimental conditions (21). The results they obtained led them to conclude that all the observed phenomena were phenomena of hypoesthesia due to the regeneration of the nerve fibers and that all forms of sensitivity tended to re-appear at about the same time in a hypoesthetic form, except sensitivity to hot, which was somewhat delayed. Their observations in regard to sensations of temperature were practically identical with Head's, but their interpretation is quite different. Normally they say there is a zone of the temperature scale in the neighborhood of the skin temperature which is indifferent and extends over about five degrees of the scale.[5] This indifference zone can be shifted up or down by warming or cooling the skin. As the distance between the skin-temperature and that of the stimulus is increased, the intensity of the sensation is increased, and it is generally necessary to have a difference of fifteen degrees before the maximum sensation is elicited. Now if there is a general reduction in the thermal sensitivity of the skin, this will produce an increase in the range of the intermediate insensibility, and a diminution in the intensity of the sensation produced by various stimulus temperatures. As an illustration: Suppose on the normal skin a stimulus of 32° is indifferent, 35° slightly warm, 38° warm, and 50° hot; if the skin is hypoesthetic, then 32° and 35° will both be indifferent, 38° faintly warm, 50° warm, while hot perhaps cannot be elicited at all. This is what really happens, according to Trotter and Davies, in the hypo-esthetic zone of the recovering area, and partly accounts for the delayed reaction to hot. Normally, a stimulus of 50° produces a sensation of hot, accompanied by a stinging quality; in the hypoesthetic area, before any thermal quality is recognizable, a stinging sensation is produced by a stimulus of 50°.

Trotter and Davies' experiment was very carefully done and seems adequate to the facts it explains, but it fails to account for the cases reported by Head where epicritic sensibility remained after the protopathic type was destroyed. There is some evidence also to support Head's theory that the two sorts of sensibility are conducted by different spinal paths. Ranson has attempted to show histologically that the fibers that serve epicritic impulses are myelinated, enter the dorsal columns of the cord from the larger ganglia of the dorsal roots, and ascend on the same side; while those that serve protopathic impulses (pain, and extremes of temperature) are unmyelinated both peripherally and centrally, enter the cord from smaller ganglia, cross immediately to the other side of the cord and ascend in the lateral columns. Temperature impulses, however, are an exception to the uncrossed epicritic tracts, and, to provide for them, a few of the myelinated fibers were found to cross (17).

The literature leaves us then, in a rather inconclusive state as regards the nature of thermal sensations and the physiological conditions of their arousal. Authorities differ, on the one hand, as to whether hot is a simple sensation or a fusion involving both warm and paradoxical cold sensations; and, if a fusion, whether it can be analyzed or not. On the other hand, they are not agreed as to whether the facts of dissociation are to be explained by assuming different sensory mechanisms for extreme and medium stimuli, or by an overlapping nerve supply, or by a greatly diminished sensitivity. More clinical evidence of a searching character is needed before positive conclusions can be drawn.

EXPERIMENTAL

STATEMENT OF PROBLEM

The problem that I here present in its preliminary results is that of devising a clinical test of temperature sensitivity that may be of some diagnostic value in certain diseases of the nervous system. The test used is a test of reaction-time to two distinctly different stimuli applied simultaneously. It was suggested by Dr. Tilney that a disturbance in the response to simultaneous stimuli of cold and hot might be apparent at an earlier stage than would appear from tests of either sensation alone, and might consequently be of early diagnostic value. This part of the experiment is concerned only with the responses of normal subjects, and the problem specifically stated becomes: What is the nature of the normal individual's reaction to two stimuli, one cold, one hot, applied simultaneously to immediately adjacent areas of the skin, in various regions of the skin surface?

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[5] This is a much wider zone than the results of any of the earlier investigators imply.

APPARATUS

The stimulus was applied by means of little blocks of soft iron, attached to wooden handles, bearing a letter or number for ease of identification. These blocks were of two sizes, to suit the areas tested—the larger ones were two inches long, one and one-half inches wide and one-quarter inch thick; the smaller, one inch by three-quarters by one-quarter. There were ten of each size.

The blocks to be heated were kept in a bed of sand in a copper drying-oven, into the roof of which a thermometer was inserted—held by a cork—which reached to the bed of sand and recorded its temperature. The oven was heated over a Bunsen burner, and could be maintained at an accuracy of two degrees Centigrade range, which was considered sufficiently accurate for the purposes of the experiment, at least in its preliminary stages. It required a good deal of attention, however, to keep it at the desired temperature. The blocks to be cooled were placed likewise on a bed of sand, in a heavy zinc box which rested on a wooden rack, beneath which broken-up ice was placed. The box was also fitted with a cork into which a thermometer was inserted, reaching to the floor of sand. The accuracy of this device likewise varied within two degrees or less.

Before the testing began, a rough attempt was made to determine how long, under the conditions of the experiment, a block would retain its heat. A hole was made in one of the blocks so that the bulb of a thermometer would fit into it, and some tests were made with this block. It was found that in a half-minute (which was as long as a block was ever kept out of the oven), of which time it was in contact with the skin from five to ten seconds, the block lost less than half a degree Centigrade, but that after returning the block to the oven it was four or five minutes before the block regained the oven-temperature. Enough blocks were provided so that no block was used oftener than once in five minutes, and only rarely oftener than once in seven or eight minutes.

It was desired for the purposes of the experiment to time the reactions in some simple fashion that would be suitable to use for clinical purposes. A stop watch could not be used, since both the experimenter's hands were occupied with the blocks; therefore a Thomas polygraph was used, so arranged that the experimenter could record the reactions with her foot. A tambour was so fixed that a light tap upon it with the experimenter's toe was transmitted to the writing lever of the instrument and made a mark upon the record paper. The polygraph was provided with a Jacquet chronograph, which marked fifths of a second. Writing pens were used, thus obviating the necessity of smoked paper, and the record could therefore be marked permanently as it was made.

MATERIAL AND METHOD

Five women acted as subjects for the experiment. They included a janitress of a flat and a college instructor; they ranged in intelligence from mediocre to superior ability, and in age from twenty-one to fifty years. They had no known nervous disturbances—except a slight neuritis reported once by one subject—although one subject was regarded as of a nervous temperament.

These five subjects were tested on forty test spots previously determined, each area being tested four times with the double stimulus—both cold and hot. An enumeration of the spots follows:

Area 1.—Fingers of right hand, dorsal surface. Area covered by two small blocks (held with the long sides juxtaposed and the long axes running in a radio-ulnar direction) on the index and middle fingers, closely peripheral to the metacarpal bones.

Area 2.—Fingers of left hand, dorsal surface. Same area.

Area 3.—Right hand, dorsal surface. Area covered by two small blocks (held in same position) immediately proximal to second and third metacarpal bones.

Area 4.—Left hand, dorsal surface. Same area.

Area 5.—Right forearm, dorsal surface. Area covered by two large blocks (held with the short sides in juxtaposition and the long axes running in a peripherocentrad direction) which were set down proximally to a line drawn in a radio-ulnar direction one inch above the protuberance of the ulna, the long axis of the blocks coinciding with a line intersecting the first at right angles, one inch radiad to the protuberance of the ulna.

Area 6.—Left forearm, dorsal surface. Same area.

Area 7.—Right arm, lateral surface. Area covered by two large blocks (held with the long sides juxtaposed and the long axis at right angles to the axis of the arm) which were set down proximally to a line drawn two inches above the olecranon process of the ulna, the short axis of the blocks coinciding with a line joining the olecranon process of the ulna and the acromion process of the scapula.

Area 8.—Left arm, lateral surface. Same area.

Area 11.—Fingers of right hand, ventral surface. Area corresponding to that of Area 1.

Area 12.—Fingers of left hand, ventral surface. Same area.

Area 13.—Right hand, palm. Area corresponding to that of Area 3.

Area 14.—Left hand, palm. Same area.

Area 15.—Right forearm, ventral surface. Area corresponding to that of Area 5, but measured from protuberance of radius.

Area 16.—Left forearm, ventral surface. Same area.

Area 17.—Right arm, mesial surface. Area covered by two large blocks (held as for Area 7) which were set down proximally to a transverse line one inch above the crease made by bending the elbow, the short axis corresponding with the line of the biceps. The latter line was measured from the inner condyle of the humerus, and the distance varied with the different subjects but remained the same for any one subject throughout the experiment.

Area 18.—Left arm, ventral surface. Same area.

Area 19.—Face, middle of forehead. Area covered by two small blocks (held with the long sides juxtaposed and the short axis in a cephalocaudad direction), set down so as just to clear the eyebrows.

Area 20.—Dorsal surface of neck. Area covered by two small blocks (held as for Area 19), set down in the median line, just below the hair.

Area 21.—Face, right cheek. Area covered by two small blocks (held as for Area 19), set down between the hair and the point of attachment of the lobe of the ear, just in front of the tragus.

Area 22.—Face, left cheek. Same area.

Area 23.—Right leg, lateral surface. Area covered by two large blocks (held with the short sides in juxtaposition and the long axis in a cephalocaudad direction) which were set down proximally to a line drawn transversely three inches above the external malleolus, the long axis of the blocks coinciding with a line intersecting the first at right angles, drawn upward from the same point.

Area 24.—Left leg, lateral surface. Same area.

Area 25.—Right leg, mesial surface. Area corresponding to that of Area 24, measured from the internal malleolus.

Area 26.—Left leg, mesial surface. Same area.

Area 27.—Right thigh, lateral surface. Area covered by two large blocks (held with the long sides juxtaposed and the short axis in a cephalocaudad direction), which were set down proximally to a line drawn transversely three inches above the outer condyle of the femur, the short axis of the blocks coinciding with a line drawn upward longitudinally from the same point.

Area 28.—Left thigh, lateral surface. Same area.

Area 29.—Right thigh, mesial surface. Area corresponding to that of Area 27, measured from two inches above the inner condyle of the femur.

Area 30.—Left thigh, mesial surface. Same area.

Area 31.—Right foot, sole. Area covered by two small blocks (held with the long sides juxtaposed and the long axis running transversely), set down at the intersection of two lines—a longitudinal line from the middle of the curve of the heel to the junction of the second and third toes, and a transverse line bisecting this.

Area 32.—Left foot, sole. Same area.

Area 31.1.—Right foot, dorsal surface. Area covered by two small blocks (held as for Area 31), set down proximally to the third and fourth toes.

Area 32.1.—Left foot, dorsal surface. Same area.

Area 33.—Abdomen, median line, caudad to navel. Area covered by two large blocks (held with the long sides juxtaposed and the long axis in a transverse direction), set down immediately caudad to the navel.

Area 34.—Abdomen, median line, immediately cephalad to navel.

Area 35.—Abdomen, right side. Area covered by two large blocks (held as for Area 33), set down at the intersection of a transverse line at the level of the navel and a longitudinal line three inches to the right of navel.

Area 36.—Abdomen, left side. Same area.

Area 37.—Back, median line. Area covered by two large blocks (held with the long sides juxtaposed and the long axis running transversely), set down caudad to a transverse line at the level of the lowest rib, the short axis of the blocks coinciding with the line of the vertebræ.

Area 38.—Back, median line. Area covered by two large blocks (held as for Area 37), set down cephalad to a transverse line four inches cephalad to the level of the lowest rib. (In the case of Subject G six inches was used instead of four.)

Area 39.—Back, right side. Area covered by two large blocks (held as for Area 37), set down at the intersection of the line at the level of the lowest rib and a longitudinal line four inches lateral to the spines of the vertebræ.

Area 40.—Back, left side. Same area.

These forty areas were tested four times for each individual, twice with the hot block above the cold (*i. e.*, proximal or cephalad to it), and twice with the hot block below the cold (*i. e.*, distal or caudad to it). The two positions are referred to as Positions A and B respectively. Twenty-six of the areas were also stimulated with two cold and two hot blocks, separately.

The experiment was divided into two series, each consisting of sixty-six stimulations, forty with the double stimulus, thirteen with hot and thirteen with cold. The series were arranged in as nearly a haphazard order as was possible. Certain limitations were necessary, however, in order to make the experiment go smoothly. It was necessary, for example, to restrict the region tested in any group of stimulations to a part of the body that could be reached when the subject was lying in one position; it was not permitted to test the same area twice without a lapse of at least three stimulations between (to avoid disturbance from after-images); the hot and cold, large and small blocks had to be so distributed that no block was used oftener than once in five minutes, etc. The series finally took form as follows:

SERIES I

Double Stimulations given in Position A

Group 1. Right Arm and Hand

TEST	AREA		BLOCKS
1	11	cold	S 1, 2
2	5	—	L a, 1
3	3	hot	S a, b
4	17	cold	L 2, 3
5	11	—	S c, 3
6	15	hot	L b, c
7	13	—	S d, 4
8	3	—	S e, 5
9	17	—	L d, 4
10	5	hot	L e, a
11	1	—	S a, 1
12	15	—	L b, 5

Group 2. Abdomen and Face

TEST	AREA		BLOCKS
13	35	—	L 1, 2
14	34	—	L c, 3
15	21	—	S b, 2
16	36	—	L d, e
17	19	—	S c, 3
18	22	hot	S d, e
19	33	—	L a, 4
20	35	—	L b, 5
21	21	hot	S a, b
22	19	cold	S 4, 5
23	36	—	L c, 1
24	22	—	S c, 1

Group 3. Legs and Feet

TEST	AREA		BLOCKS
25	25	—	L d, 2
26	28	—	L e, 3
27	30	—	L a, 4
28	24	—	L b, 5
29	31	hot	S d, e
30	32.1	—	S a, 2
31	23	—	L c, 1
32	32	—	S b, 3
33	29	cold	L 2, 3
34	26	—	L d, 4
35	27	—	L e, 5
36	31.1	—	S c, 4
37	32	cold	S 5, 1
38	31	—	S d, 2
39	24	hot	L a, b
40	23	cold	L 1, 2
41	29	—	L c, 3
42	30	hot	L d, e

Group 4. Back, Neck, Lateral Surface of Arms

TEST	AREA		BLOCKS
43	39	—	L a, 4
44	40	cold	L 5, 1
45	8	—	L b, 2
46	38	—	L c, 3
47	7	hot	L d, e
48	20	—	S d, 4
49	37	—	L a, 4
50	8	cold	L 5, 1
51	40	—	L b, 2
52	7	—	L c, 3
53	39	hot	L d, e
54	20	cold	S 5, 1

Series I was given to three subjects on the first and third days, and to two on the second and fourth; while Series II was given to the former on the second and fourth days and to the latter on the first and third. Practice tests were given to each subject before each group, on the areas included in the group, and the arrangement of the series was such as to distribute as evenly as possible the effects of practice.

Group 5. Left Arm and Hand

TEST	AREA		BLOCKS
55	16	cold	L 4, 5
56	18	—	L a, 1
57	4	—	S a, 2
58	6	cold	L 2, 3
59	2	—	S b, 3
60	12	—	S c, 4
61	14	—	S d, 5
62	18	hot	L b, c
63	4	hot	S e, a
64	16	—	L d, 4
65	12	cold	S 1, 2
66	6	—	L e, 5

SERIES II

Double Stimulations given in Position B

Group 1. Left Arm and Hand

TEST	AREA		BLOCKS
1	6	—	L a, 1
2	18	—	L b, 2
3	16	hot	L c, d
4	12	—	S a, 1
5	2	—	S b, 2
6	4	cold	S 3, 4
7	14	—	S c, 5
8	18	cold	L 3, 4
9	12	hot	S d, e
10	16	—	L e, 5
11	6	hot	L a, b
12	4	—	S a, 1

Group 2. Back, Neck, Lateral Surfaces of Arms

TEST	AREA		BLOCKS
13	40	hot	L c, d
14	8	—	L e, 1
15	38	—	L a, 2
16	7	cold	L 3, 4
17	39	—	L b, 5
18	20	hot	S b, c
19	8	hot	L c, d
20	40	—	L e, 1
21	37	—	L a, 2
22	39	cold	L 3, 4
23	7	—	L b, 5
24	20	—	S d, 2

Group 3. Legs and Feet

TEST	AREA		BLOCKS
25	29	—	L c, 1
26	23	—	L d, 2
27	31	cold	S 3, 4
28	32	hot	S e, a
29	30	cold	L 3, 4
30	26	—	L e, 5
31	29	hot	L a, b
32	32.1	—	S b, 5
33	27	—	L c, 1
34	25	—	L d, 2
35	31.1	—	S c, 1
36	31	—	S d, 2
37	24	cold	L 3, 4
38	32	—	S e, 3
39	28	—	L e, 5
40	23	hot	L a, b
41	30	—	L c, 1
42	24	—	L d, 2

Group 4. Abdomen and Face

TEST	AREA		BLOCKS
43	35	hot	L e, a
44	19	—	S e, 3
45	33	—	L b, 3
46	21	—	S a, 4
47	36	—	L c, 4
48	22	cold	S 5, 1
49	34	—	L d, 5
50	19	hot	S b, c
51	35	—	L e, 1
52	21	cold	S 2, 3
53	36	cold	L 2, 3
54	22	—	S d, 4

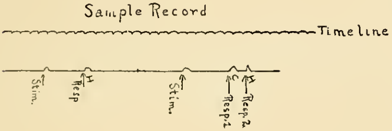
Group 5. Right Arm and Hand

TEST	AREA		BLOCKS
55	13	—	S e, 5
56	15	cold	L 4, 5
57	3	—	S a, 1
58	17	hot	L a, b
59	1	—	S b, 2
60	11	—	S c, 3
61	15	—	L c, 1
62	5	—	L d, 2
63	3	cold	S 4, 5
64	17	—	L e, 3
65	11	hot	S d, e
66	5	cold	L 4, 5

The temperature of the hot stimulus was 40°–42° C.; that of the cold stimulus was 18°–20°. A few variations beyond these limits are noted in the individual records of Table 1. The room temperature varied from 21° to 24°. This temperature, somewhat higher than ordinary room temperature, was chosen because it was desired to keep the part of the body tested exposed. However, even this temperature was not comfortable for most subjects, and the covered parts of the body were not generally exposed for more than five minutes before they were tested. They were kept uncovered throughout the test of any region.

PROCEDURE

The subject lay on a cot in such a position that the experimenter, standing beside her with one foot over the tambour used to record the reaction, could, with very little movement, take the blocks from their resting places and set them down on the part to be tested. The subject was instructed somewhat as follows: “I am going to perform an experiment to see how you respond to hot and cold. When I put this little block down on your hand, I want you to tell me just as quickly as you can what you feel. Try it now” . . . (Several trials, either stimulus.) “Now I'll put both of them on you, and you tell me which you feel first, just as soon as you feel it; and then tell me just as soon as you feel the other. Close your eyes. Ready, on your arm!” Several trials were given, until S understood that she was to respond as soon as she felt anything. Some practice was required before S was able to respond with certainty on the first day, but thereafter there was rarely any difficulty. The directions were summed up, after S felt fairly sure of her replies: “Now you see what I am going to do. Sometimes you will feel both, and sometimes you will feel only one or the other, so you must pay close attention and answer what you feel just as soon as you feel anything.” S then closed her eyes, the area to be tested was named; E removed the blocks from their boxes, started the polygraph, gave a ready signal, and set the blocks down, simultaneously tapping with her foot upon the tambour which operated the writing lever of the polygraph. S's responses were likewise recorded by a tap of E's foot, the blocks were returned to their boxes, the number of the test and S's verbal response (whether “cold-hot,” “hot-cold,” “cold,” or "hot") were noted beside the drum record of the reaction-time, and the second area was then tested in the same way. A sample of the record paper is reproduced below.



If the double stimulus was given and both cold and hot were reported, the stimulus was immediately removed. If both were given and only one reported, the stimulus was left in contact about five seconds and then removed. This same procedure was followed when only one stimulus was given, so that S's replies could not be controlled by knowledge of her errors.

If for any reason the record for any test was unsatisfactory—if the ink flowed too freely and blurred, if the tambour failed to record, if E was conscious of having bungled and given the stimulus badly, as occasionally happened—the test was repeated at the end of the group in which it occurred, without S's knowledge of its being a repetition.

When the record was completed, it was scored by counting the time marks between the moment of stimulation and the first response, and between the latter and the second response. To do this, lines were ruled from the rise of the recording mark to the time line by means of a straight edge. Readings were taken to the nearest tenth of a second.

CONDITIONS AFFECTING RESULTS

The reaction-time to cold has generally been found to be quicker than that to hot—a fact that corresponds to the probably deeper situation of the warm end-organs below the skin surface. It would therefore be assumed that if both stimuli were applied at the same time, other things being equal, the reaction to cold would precede that to hot. My results show that in most cases this is what happens (*v.* Table 1), but the majority is in some cases so slight that it is important to consider the "other things."

Limitations of Method.—In the first place, the limitations of the method must be taken into account. The method of measuring the reaction itself has a considerable possibility of error. The simultaneity with which E actually sets down the blocks and taps with her toe upon the recording tambour may be questioned. The error might be quite large in this case, for it is a fairly complicated procedure; nevertheless, I think the general consistency of each individual's responses (which may be seen from a glance at Figure 4) justifies the assumption that as a matter of fact this error was not large enough to affect the results materially. Then the recording of the subject's responses by E is also subject to a slight variable error; but this would undoubtedly be less—since E was practised before she began the tests—than the error of unpractised subjects in recording their own responses by unfamiliar means. The method of reading the record is also susceptible of a certain amount of error, which would not be greater in any case than one-tenth of a second. A more serious source of error, however, in the judgment of the experimenter, is the possibility of a slight inaccuracy in setting the blocks down exactly together and with equal pressure. An attempt was made to control this error by providing a tambour to be attached to the blocks, which would trace on the record the moment at which each block made contact with the skin. This device could not, however, be made to work satisfactorily. Consequently when the experimenter recognized that she had made a distinctly bad stimulation, she sometimes repeated it to see what the result would be. Sometimes also when the response was given in the order "hot-cold," the experimenter repeated the stimulus even though she thought it had been given accurately. This was not done systematically because it would have lengthened the experiment too much; but the following is a summary of the total number of times a test was repeated which first gave the response "hot-cold."

First Response "Hot-Cold"

	1st Badly Given		*1st Not Badly Given*	
	2nd Response		2nd Response	
Subject	C—H	H—C	C—H	H—C
G.	7	—	1	—
Co.*	1	1	13	8
K.	—	—	—	—
H.†	1	—	4	1
Cl.	—	2	6	5

* One response was "cold" only. In one test that gave the response H—C, E thought that the cold block touched first, but this test was not repeated.

† One response was "hot" only.

Of twelve tests badly given and repeated, nine gave the response C–H, and three H–C on the repetition. Of thirty-eight tests repeated, though not consciously badly given, twenty-four gave the response C–H, and fourteen H–C on the repetition. This would seem to indicate that while some of the responses that occurred in the order H–C may have been due to inaccuracies of method of stimulation, some of them probably were not. This source of error, however, remains undetermined and probably considerable.

Changes in Temperature of Skin or Stimulus.—In the second place the relation of the skin-temperature to the stimulus-temperature, and also the actual stimulus-temperature, may have affected the reaction-time in a variable way. Although the room temperature was fairly constant, the skin-temperature of different individuals on different days, on different regions of the body, probably varied rather widely. This is very likely one reason for the greater constancy of the reactions on the abdomen and back, which is shown by every figure—a less scattered distribution on these areas for each individual, a smaller variation among different individuals, and a higher percentage of responses in the order *cold-hot*. For, of course, the skin-temperature of the usually covered areas of the body varies less than that of the extremities and uncovered areas. The thighs and the back of the neck also show a greater constancy of response than the hands and feet. Variability of skin-temperature, then, will account for some of the variability of reaction.

The variations in the temperature of the stimulus constitute a possible source of error more difficult to determine. The hot stimulus varied for the most part between 40° and 42° C. On three days, a few tests were made with the oven temperature at 42.5°, one day it rose to 43°, and one day fell to 38°; but if the individual records for those days are examined, it does not appear that the general character of the results was affected (*v.* Table 1). The cold temperature varied between 18° and 20°, except on two occasions—once when it was between 18° and 16°, and once between 17° and 19°.

The hot stimulus temperature was chosen because it was desired to have a temperature distinctly hot, yet below the threshold for burning. This point of course varies; Neumann places it on the average at 43.5°, Head at about 50°. Temperatures below 40° frequently do not elicit a hot sensation. Therefore the limits were so set. In a similar way, a cold, distinctly cold but not painful, was desired. Crawford places the limit for great intensity of cold at 15°, and a temperature above 20° is not distinctly cold to a part of the skin whose temperature is 27°—as that of hands and feet often is. Therefore a temperature of 18°–19° was considered best.

The reaction-time depends, to some extent, on the temperature of the stimulus. Goldscheider found that for a limited area of the forearm, the mean reaction-time to cold for a stimulus of 20°–19° was .377 seconds; and for 15.5°–15°, .278 seconds—a difference of a tenth of a second for a range of four degrees. He also found that for a stimulus of 40°, the mean time was .777 seconds; and for 44°, .585 seconds—a difference slightly less than two-tenths of a second for a range of four degrees. Thus the variations in the stimulus temperature may have affected the results by a few hundredths of a second, but not significantly.

Topography of Warm and Cold Spots.—In the third place, the number of warm and cold spots in the areas tested may have been unequal, and have given a different emphasis to one sensation or the other. This effect should have been neutralized by the procedure of reversing the relative position of the blocks in alternate experiments. Nevertheless, slight discrepancies in the exact location of the spots might perhaps have thrown a strongly reacting spot into one-half or the other of any area, or one-half or the other may have contained a particularly sensitive spot of one kind. This possible source of error could not be corrected without a laborious exploration of the skin of every subject, which would not be worth while. It might perhaps be inferred that some idiosyncrasy of warm or cold spots is responsible for those occasional instances when one area gave a hot-cold response in each of the four tests, or only one reaction to each test. Examples of this may be seen in Table 1: Subject Co., Areas 11, 14, 16; Subject H, Area 31. It may be noted that Area 13 in three subjects gave three responses in the order *hot-cold.*

These, then, are the factors that are or may be present in the results and should be borne in mind.

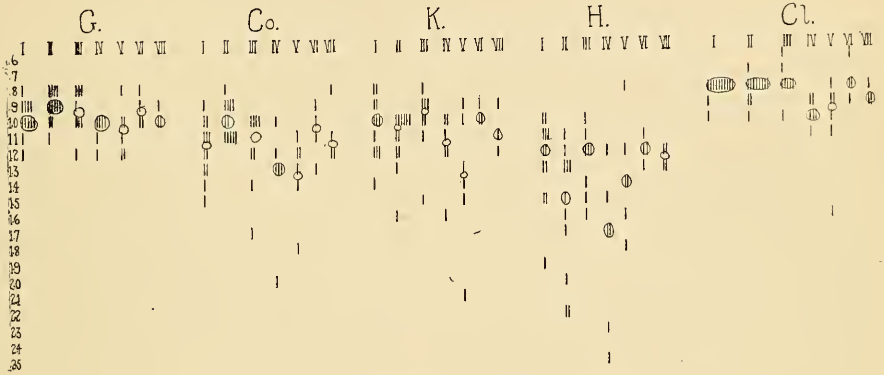


FIG. 1. DISTRIBUTION OF REACTIONS TO SINGLE STIMULI—COLD

Numerals I–VII indicate regions in order as follows: right arm, left arm, head, right leg, left leg, abdomen, back. Each stroke indicates time for one reaction on the region under which it occurs. Circles indicate medians for each region.

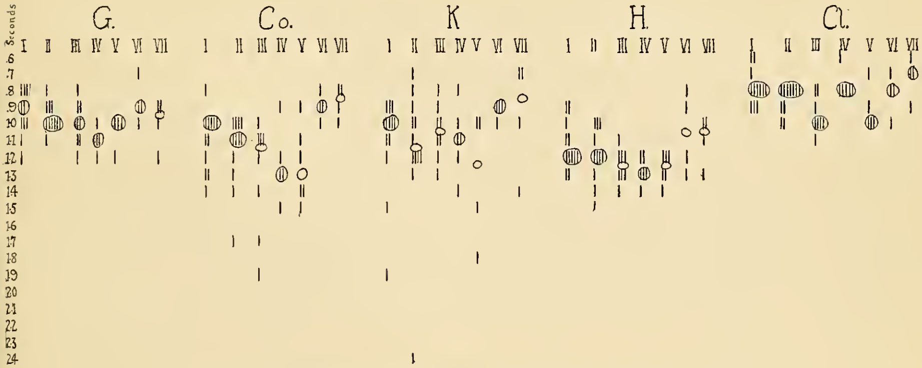


FIG. 2. DISTRIBUTION OF REACTIONS TO SINGLE STIMULI—HOT

TABLE I

Reaction-Time (in Tenths of a Second) for all Double Stimulations

Subject G

Position of stimuli: Pos. A, Experiment 1 and 3; Pos. B, Experiment 2 and 4
Temperature: H—Ex. 1, 42°–39.5° C.; Ex. 2, 40–42; Ex. 3, 41–42.5; Ex. 4, 40–42
C—Ex. 1, 18°–19°; Ex. 2, 18–19; Ex. 3, 18–19; Ex. 4, 18–20
Room—Ex. 1, 21–23°; Ex. 2, 23–24; Ex. 3, 24; Ex. 4, 21–23
Dates of experiment: March 15, April 12, April 18, May 24, 1917

Area	*Order C–H*								*Order H–C*								*Single Resp.*			
	1st Intv.				*2nd Intv.*				*1st Intv.*				*2nd Intv.*							
Ex.	*1*	*2*	*3*	*4*	*1*	*2*	*3*	*4*	*1*	*2*	*3*	*4*	*1*	*2*	*3*	*4*	*1*	*2*	*3*	*4*
1	10	10	12	8	5	4	3	3												
3	9	10	10	8	6	5	4	4												
5	10	12	9	9	4	5	4	4												
7	12	10	9	8	4	4	4	3												
11	18		11	10	4		4	4										C13		
13	10	10	10″	9	4	3	4″	3			14′				4′					
15	9	9	10	8	3	4	4	3												
17	11	9	9	9	4	4	5	3												
2	13	10	11	9″	5	3	4	3″				11′				3′				
4	12	12	10″	10	4	3	4″	4			9′				3′					
6	12	9	12	10	4	3	4	3												
8	10	9	9	8	4	4	4	4												
12		9	11	8		6	3	4	13				4							
14		11	9″	9		4	4″	4	11		9′		4		3′					
16	9	10	9	8	4	3	4	3												
18		9	8	10		4	3	3	9				6							
19		9		8		4		4									C10	C11		
21	10	8	10	8	4	7	4	4												
22	10	10		8	4	4		7											C9	
20		10	10	11		6	4	7	14				4							
23	10			8	11			7		12				3					C10	
25	13	10	10	8	4	4	4	6												
27	11	10	8	9	5	4	3	4												
29	10	9	8	9	5	4	5	4												
31	13	14	10	11	4	4	6	5												
31.1		11	9	10		21	4	6									H12			
24	8		8	8	13		4	5		12				4						
26	10	10	8	9	4	7	5	4												
28	9	10	8	8	4	4	4	5												
30	10	10	19	8	3	4	4	3												
32	15	10	10	10	5	4	10	4												
32.1	9	14	9	11	4	4	7	6												
33	9	9	8	8	4	4	4	4												
34	10	10	12	8	4	3	4	5												
35	9	8	9	9	4	4	4	3												
36	10	9	10	8	5	4	3	3												
37		10	11	8		4	4	4	8				4							
38	13	8	10	8	4	3	4	3												
39		12	10	8		4	4	3	9				3							
40		11	9	10		7	4	3	8				4							

TABLE I (*continued*)

Reaction-Time for Double Stimulations

Subject Co.

Position of stimuli: Pos. A, Experiment 1 and 3; Pos. B, Experiment 2 and 4
Temperature: H—Ex. 1, 41°–41.5° C.; Ex. 2, 41–41.5; Ex. 3, 40–42; Ex. 4, 40–42
C—Ex. 1, 18–18.5°; Ex. 2, 18; Ex. 3, 18–20; Ex. 4, 18–20
Room—Ex. 1, 24°–22°; Ex. 2, 22–23; Ex. 3, 21–22; Ex. 4, 21–22
Dates of experiment: March 12, April 17, May 18, May 22, 1917

Area Ex.	*Order C–H*								*Order H–C*								*Single Resp.*			
	1st Intv.				*2nd Intv.*				*1st Intv.*				*2nd Intv.*							
	1	2	3	4	1	2	3	4	1	2	3	4	1	2	3	4	1	2	3	4
1	16	12	10	11	3	4	5	3												
3			15″				$\underline{21''}$					10				4	C13	H13	C13′	
5	11				6					14	10	13*		4	4	4*				
7	11		10	11″	4		4	4″		11		14′		4		4′				
11									18	17	12*	10*	4	5	4*	4*				
13		11	14″			4	4″		15		16′	10	8		4′	4				
15			10	10			4	4	14	11			6	4						
17									15	10	11	10	11	4	4	4				
2		8	13″	11		4	4″	3	13		10′		10		4′					
4		10	14			4	4					21				8	C26			
6		10	12			5	4		14			11	5			6				
8	10	11	11″		5	6	4″				12′	10*			5′	4*				
12		9		11		4		4	11		12*		6		4*					
14									12	10	10*	10	11	4	4*	4				
16				12″				4″	13	11	11	12′	6	4	5	4′				
18			10″	10			4″	3	12	10	8′		7	12	4′					
19			11	10			4	4		12				4			C21			
21	13		14	10	6		4	5		11				4						
22	11		11″	9	6		4″	4		15	11′			9	4′					
20	17	14			5	5					16	10			12	4				
23	14		10	10	6		4	10		†										
25	14	15	13	11	6	6	12	6												
27	10	14	12	11	5	4	4	4												
29	10	11	11″	9	5	6	4″	4			12′				4′					
31	15				12													C21	C12	C16
31.1	13	15	14		12	6	9													C20
24			13	†			4										C24	C20		
26	14	15	17	14	6	4	4	4												
28	14	13	11″	9	6	4	5″	3			12′				4′					
30			10″	10			4″	4	12	15	10′		6	9	4′					
32		12					4		†			16				18		C13		
32.1												15*				24*	C15	H22	C14	
33		10		10		4		6	18	13			7	14						
34	19		12″	10	4		4″	4		13	12′			6	6′					
35	13	12		9	8	4		3		10				4						
36	17		10	9	6		4	3		16				4						
37	10	11	9	10	5	3	4	3												
38	10	10	9	12	6	5	4	3												
39	12		8″	9	6		6″	4		11	11′			7	4′					
40		11	10	8		5	5	3	9				6							

* Stimulus was repeated and gave reaction in the same order. † Judgment uncertain or confused.
Figures underlined indicate a reaction of "warm" instead of "hot."

TABLE I (*continued*)

REACTION-TIME FOR DOUBLE STIMULATIONS

Subject K

Position of stimuli: Pos. A, Experiment 1 and 3; Pos. B, Experiment 2 and 4
Temperature: H—Ex. 1, 41°–42° C.; Ex. 2, 41.5–43; Ex. 3, 43–40; Ex. 4, 40.5–42
C—Ex. 1, 18°–19°; Ex. 2, 18–18.5; Ex. 3, 18–19; Ex. 4, 18–18.5
Room—Ex. 1, 23°; Ex. 2, 22–23; Ex. 3, 23–24; Ex. 4, 23
Dates of experiment: March 19, March 27, April 2, April 3, 1917

	Order C–H								*Order H–C*											
Area	*1st Intv.*				*2nd Intv.*				*1st Intv.*				*2nd Intv.*				*Single Resp.*			
Ex.	*1*	*2*	*3*	*4*	*1*	*2*	*3*	*4*	*1*	*2*	*3*	*4*	*1*	*2*	*3*	*4*	*1*	*2*	*3*	*4*
1	14		10	12	4		3	4		10				6						
3	16		18		5		3			13				4						
5	13		15		3		3			9		10		5		3				
7				8				4	22	21	14		6	7·	3					
11		10	11			6	3		9				5							B20‡
13				†					18	12	12	†	5	5	3					
15	14				5					10		11		4		4			B13	
17	11	12		8	5	6		4											B19	
2			10	11			3	3	12				4					H13		
4		14				6			16		14	14	5	4	4					
6	10				4						9	10			4	4		B13		
8	10		9	8	4		4	7		10				4						
12	12	11	19		5	4	4					13				6				
14			10	14			4	4	12				4						B17	
16		12				4			21				4					B12	C11	
18	14			12	4			4		9	14			3	4					
19		11		10		3		3	11		8		3		3					
21		10	9	9		4	4	3	16				5							
22	14		10	12	5		4	4		11				5						
20	13		12	9	4		4	3										B30		
23			11				4		12			12	4		4			B29		
25	10		18		6		3			14		11″		6		3″				B14′
27	11	11			4	4						9				3			C8	
29	13	9	8	12	5	4	4	3												
31			18				4		28	18			5	6						B22
31.1	17	10		13	5	16		8											H19	
24	21				5					16		18		4		4			B28	
26									22	9		10	4	4		4			B18	
28	11	10	9	11	6	4	8	4												
30	16	9	8	12	8	4	6	4												
32		12	12			4	15		13			14	9			4				
32.1	14		11	11	5		7	6										H22		
33	11	12.	7	10	4	7	4	5												
34	11	10		10	7	3		5			8				3					
35	10	10	10		4	3	3					9				4				
36	11	10	9	10.	4	4	4	4												
37		11		11		5		4	13				5							B37
38	11	9	10	9	4	4	3	4												
39	13	10	8	10	3	4	4	4												
40	10	9		10	4	6		4			10				6					

† No record—test omitted.

‡ Reactions preceded by *B* are cases in which subject declared the sensations to be perceived simultaneously.

TABLE I (*continued*)

Reaction-Time for Double Stimulations

Subject H

Position of stimuli: Pos. A, Experiment 2 and 4; Pos. B, Experiment 1 and 3
Temperature: H—Ex. 1, 38°–41° C.; Ex. 2, 40–42; Ex. 3, 40–42; Ex. 4, 40–42.5
C—Ex. 1, 18–16°; Ex. 2, 17–17.5; Ex. 3, 17–19; Ex. 4, 18–20
Room—Ex. 1, 22–23°; Ex. 2, 22; Ex. 3, 21–23; Ex. 4, 20–22
Dates of experiment: March 13, April 10, May 3, May 15, 1917

Area	*Order C–H*								*Order H–C*								*Single Resp.*			
	1st Intv.				*2nd Intv.*				*1st Intv.*				*2nd Intv.*							
Ex.	*1*	*2*	*3*	*4*	*1*	*2*	*3*	*4*	*1*	*2*	*3*	*4*	*1*	*2*	*3*	*4*	*1*	*2*	*3*	*4*
1		13		14		11		31	12				6						H17	
3	13	12			7	12					14				6					C15
5		12				4					15	14			9	19	H18			
7		15		13		21		5			14				13		H28			
11	18		16		6		4			14				3						H14
13				16				14	14	12	14		10	11	4					
15		10				4						15				30	H13		H15	
17		11				4			15		13		6		13					H12*
2	16				5						19				21			H13		H16
4	26		13	16	18		12	12										H14		
6			11	14			7	9	22	13			5	5						
8	14	12	13	14‴	5	4	7	6‴				17′				10′				H15″
12	12		12		8		15			14		17		22		7				
14	15	12			6	5					18				12					C15
16	12	12		13	4	7		8			15				10					
18			14	14			4	6		14				5			H13			
19	14	14		18	7	6		19			14				17					
21	10		21	20	6		4	19		14				8						
22		11		14		6		14			15				6		H14			
20	13	12	14	13	7	8	5	11												
23				14				8	14		16		4		9				H16	
25	15	15	13		8	5	6													H18
27				18				22									C13	C14	H16	
29	13	13	13		6	5	6					16				17				
31																	C12	C15	C18	C16
31.1	17	21		17	4	26		7											H19	
24	12	14	14	14	9	6	13	8												
26	16	19	17	19	5	4	6	5												
28	13	12	17		5	6	5													C12
30	12	12			5	6					15				12				H20	
32		14				28											C11	C15		C14
32.1		16	15	14		20	6	10									H18			
33		12		11		4		9			16				5		H16			
34		11	13	11″		5	5	6″				18′				5′	H22			
35		14	17			7	6					14				14	H16			
36	12	11	14	14	5	4	4	5												
37	11	12	12	11	6	4	4	5												
38	10	12	11	18″	5	5	7	6″				10′				4′				
39	10	12	11	14″	4	5	5	6″				15′				5′				
40	13		11	13	5		4	24		12				8						

TABLE I (*continued*)

REACTION-TIME FOR DOUBLE STIMULATIONS

Subject Cl.

Position of stimuli: Pos. A, Experiment 2 and 4; Pos. B, Experiment 1 and 3
Temperature: H—Ex. 1, 40.5°–42° C.; Ex. 2, 40.5–42; Ex. 3, 40–42.5; Ex. 4, 40–42
C—Ex. 1, 17.8°–19°; Ex. 2, 18–20; Ex. 3, 18–20; Ex. 4, 18–20
Room—Ex. 1, 22°–23°; Ex. 2, 24–25; Ex. 3, 21–22; Ex. 4, 21–22
Dates of experiment: March 26, April 23, May 23 (*a. m.*), May 23 (*p. m.*), 1917

Area	*Order C–H*								*Order H–C*											
	1st Intv.				*2nd Intv.*				*1st Intv.*				*2nd Intv.*				*Single Resp.*			
Ex.	*1*	*2*	*3*	*4*	*1*	*2*	*3*	*4*	*1*	*2*	*3*	*4*	*1*	*2*	*3*	*4*	*1*	*2*	*3*	*4*
1	9	7	11		4	3	3					11				7				
3	9	9			5	3					12	9			24	4				
5	7	7″			5	3″				9′	9	8		5′	4	3				
7			9	8			5	7	6				11					C7		
11	12	7″			10	3″				7′	12	10*		7′	4	3*				
13	14	8	9″		4	4	4″				†′	10*			†′	4*				
15		6	9	8		4	4	3									H8			
17	8	6	8	8	4	3	4	3												
2		11″	8			4″	3			10′		8*		4′		8*	H8			
4	10				6					9	9*	8		6	4*	6				
6			8				3		8	9		8	5	6		6				
8	7	9	8	7	4	4	4	6												
12		8	8			4	5					8				4	C8			
14	10		10	11	6		8	4		9*				4*						
16		9		9		4		4	8		8		5		6					
18		10	8	8		4	4	4	10				5							
19	10	8			11	4					10	7			4	4				
21	8	9	10	9	9	3	4	4												
22		9				7						8				6	C8		C12	
20	9	8	9	8	5	6	5	4												
23	12	8″	9	8	5	8″	6	15		8′				5′						
24	10	8	10	10	16	10	8	6												
27	9	8	9	6	4	5	5	9												
29	8	8	8	8	4	5	4	4												
31	9	11	9		9	9	11													C12
31.1		8	8	10		15	12	14	16				4							
24		7	9			16	7		9				4							C8
26		9		9		14		10									C10		C8	
28	10	7	8	8	5	6	6	4												
30	10	6	9	8	4	4	6	4												
32		9	12	12		11	16	11	15				14							
32.1	9	8	9	10	11	21	28	12												
33	8		10	7	3		4	3		9				4						
34	9	8	10	8	5	4	4	4												
35	8	8	9	8	4	3	4	4												
36	8	8	10	8	4	4	6	4												
37	8	9	8	8	4	4	7	5												
38		8	8	7		6	12	5	8				4							
39	7	8	9	8	4	4	5	5					5							
40		8	8	9		4	9	3	9											

† Record could not be read, but response was in Order H—C.

* Stimulus was repeated and gave reaction in the same order.

Explanation: Meaning of "Position of Stimulus" is explained under *Method.* Under "Order C–H" are grouped those reactions that gave response to *cold* first; under "Order H–C," those that gave response to *hot* first; under "Single Resp." those that gave either *cold* or *hot* alone, each figure designated by the appropriate letter. "1st Intv." means time from stimulus to first response; "2nd Intv.," time from first response to second response. Twice-accented figures indicate repetitions of the stimulus, to which the first response is shown by once-accented figures. Conditions under which repetitions were made are described under *Method.*

TABLE 2

Reaction-Time (in Tenths of a Second) for Single Stimulations

Cold

Subject *Area*	*G.*		*Co.*		*K.*		*H.*		*Cl.*		*Av. M. V.* *Seconds*	
3	11	10	13	12	10	19	9	12	8	9	1.04	.21
4	10	10	10	10	11	12	C14 H8	11	9	10	1.03	.07
11	8	9	8	11	11	9	12	12	8	8	.96	.15
12	10	9	11	11	12	10	12	13	8	8	1.04	.14
5	12	8	14	13	10	15	12	11	8	9	1.12	.20
6	12	10	14	11	12	24	12	14	10	7	1.28	.26
7	8	8	10	10	12	11	9	13	7	8	.96	.16
8	10	10	10	12	13	8	10	12	8	8	1.01	.13
15	10	9	10	10	9	10	10	12	8	9	.97	.08
16	11	9	17	13	10	12	10	15	8	8	1.13	.24
17	10	10	11	10	10	9	11	13	6	6	.96	.16
18	9	8	10	11	9	7	12	10	8	9	.93	.12
19	11	10	19	11	10	8	12	13	8	10	1.12	.20
21	10	9	11	10	10	11	14	13	11	10	1.09	.11
22	11	8	17	11	13	12	12	12	10	9	1.15	.16
20	9	12	12	14	11	9	11	13	10	9	1.10	.14
23	11	11	15	C11 H16	14	11	13	13	8	8	1.15	.20
24	C11 H4	10	12	14	C27 H6	C12 H9	12	13	10	9	1.14	.15
29	C11 H6	C8 H5	12	9	10	8	12	14	6	8	.98	.21
30	10	10	9	11	10	10	14	12	7	8	1.01	.13
31	12	10	13	13	12	11	12	13	8	8	1.12	.16
32	12	C9 H4	14	15	18	15	12	13	10	10	1.32	.20
35	10	9	8	10	10	9	9	12	8	7	.92	.11
36	9	C8 H4	9	9	9	9	8	13	10	8	.94	.10
39	10	9	8	10	7	10	10	11	6	7	.88	.14
40	12	9	8	9	14	17	13	10	7	9	1.08	.26

TABLE 2 (*continued*)

Reaction-Time for Single Stimulations

Hot

Subject *Area*	*G.*		*Co.*		*K.*		*H.*		*Cl.*		*Av. M. V.* *Seconds*	
3	10	10	14	9	12	12	12	13	8	8	1.08	.18
4	9	10	11	11	13	12	17	16	8	8	1.06	.24
11	9	10	12	10	10	9	15	19	8	8	1.10	.26
12	9	9	9	9	12	11	12	20	8	8	1.07	.24
5	9	11	15	12	12	11	10	15	8	8	1.11	.19
6	9	8	11	10	10	10	22	13	10	8	1.11	.25
7	10	9	13	11	9	10	11	11	9	8	1.01	.11
8	11	8	11	11	16	11	13	13	7	9	1.10	.18
15	12	9	11	13	14	8	11	13	8	8	1.07	.20
16	8	8	8	9	10	10	H13 C6	15	9	8	.94	.19
17	10	8	11	10	10	8	10	12	10	8	.97	.11
18	9	10	10	9	10	10	11	22	8	8	1.07	.22
19	10	8	10	10	8	11	11	15	10	8	1.01	.13
21	8	9	10	10	10	9	10	12	8	8	.94	.09
22	12	10	17	14	9	15	12	14	8	7	1.18	.25
20	10	8	12	12	9	10	16	12	6	8	1.03	.22
23	10	10	13	13	10	11	15	23	10	10	1.25	.28
24	12	11	18	12	21	9	14	14	16	10	1.37	.28
29	10	12	13	10	12	10	17	12	9	9	1.14	.18
30	10	8	13	11	13	10	12	16	9	8	1.10	.20
31	10	11	20	12	12	16	25	17	10	11	1.44	.48
32	12	10	12	14	15	14	8	18	9	11	1.23	.23
35	10	8	11	10	10	10	11	12	9	8	.99	.09
36	9	10	13	9	H12 C8	9	12	13	8	6	.99	.19
39	H8 C4	10	12	12	H13 C6	9	12	12	8	9	1.05	.15
40	10	9	11	8	11	13	13	13	H10 C8	9	1.08	.16

TABLE 3

Proportion of Responses in Order, C–H and H–C

Cold—Hot

Subject	*G.*		*Co.*		*K.*		*H.*		*Cl.*			
	No.	*%*	*No.*	*%*	*No.*	*%*	*No.*	*%*	*No.*	*%*	*Av. %*	*M.V.*
Right arm	31	94	13	34	14	45	12	38	21	57	53.6	17.6
Left arm	28	83	15	38	15	47	19	56	18	50	54.8	11.8
Head	12	75	10	59	11	62	12	75	11	69	68.0	6.0
Right leg	21	88	19	80	13	52	11	46	22	88	70.8	17.4
Left leg	23	96	12	48	14	59	17	71	19	80	70.8	14.0
Abdomen	16	100	11	65	14	88	11	64	15	94	82.2	14.6
Back	13	81	14	82	13	81	15	83	14	87	82.8	1.8
Average		88.1		55.3		62		61.9		75	69	

TABLE 3 (*continued*)

Hot—Cold

Subject	*G.* No.	*G.* %	*Co.* No.	*Co.* %	*K.* No.	*K.* %	*H.* No.	*H.* %	*Cl.* No.	*Cl.* %	*Av. %*	*M.V.*
Right arm	1	3	22	58	13	42	12	38	14	38	34.8	13.0
Left arm	6	17	23	59	12	38	9	27	16	44	37.0	12.0
Head	1	6	6	35	4	25	3	19	3	19	20.8	7.4
Right leg	1	4	1	4	7	28	3	13	2	8	11.4	7.3
Left leg	1	4	7	28	7	29	1	4	2	8	14.6	11.1
Abdomen	0	0	6	35	2	12	3	18	1	6	14.2	9.8
Back	3	19	3	18	2	12	3	17	2	13	15.8	2.6
Average		7.6		33.8		26.6		19.4		19.4	21.4	

TABLE 4

SINGLE RESPONSES TO DOUBLE STIMULI

Subject	*G.* C	*G.* H	*Co.* C	*Co.* H	*K.* C	*K.* H	*H.* C	*H.* H	*Cl.* C	*Cl.* H	*Totals* C	*Totals* H
Fingers	1	—	—	—	—	1	—	4	1	1	2	6
Hand	—	—	3	1	—	—	2	1	—	—	5	2
Arm	—	—	—	—	1	—	—	8	1	1	2	9
Face	3	—	1	—	—	—	—	1	2	—	6	1
Back of neck	—	—	—	—	—	—	—	—	—	—	—	—
Leg	1	—	2	—	—	—	—	2	3	—	6	2
Thigh	—	—	—	—	1	—	3	2	—	—	4	2
Foot	—	1	7	1	—	2	7	2	1	—	15	6
Abdomen	—	—	—	—	—	—	—	3	—	—	—	3
Back	—	—	—	—	—	—	—	—	—	—	—	—
Totals	5	1	13	2	2	3	12	23	8	2	40	31

TABLE 5

MEDIAN REACTION-TIME FOR SINGLE STIMULI—GROUPED BY SUBJECTS

Cold

Subject	*G.*			*Co.*			*K.*			*H.*			*Cl.*		
Region	*Med.*	*No. of Cases*	*P.E.*	*Med.*	*No. of Cases*	*P.E.*	*Med.*	*No. of Cases*	*P.E.*	*Med.*	*No. of Cases*	*P.E.*	*Med.*	*No. of Cases*	*P.E.*
Right arm	.9	11	.10	1.05	12	.10	1.0	12	.05	1.2	12	.05	.8	12	.00
Left arm	1.0	12	.05	1.1	12	.10	1.15	12	.10	1.2	11	.10	.8	11	.05
Head	1.0	8	.10	1.15	8	.15	1.05	8	.05	1.25	8	.05	1.0	8	.05
Right leg	1.1	4	.00	1.3	5	.05	1.1	6	.10	1.3	6	.05	.8	6	.00
Left leg	1.0	4	.00	1.3	6	.15	1.25	4	.25	1.25	6	.05	1.0	5	.05
Abdomen	.9	4	.00	.9	4	.00	.9	4	.00	1.05	4	.15	.8	4	.00
Back	.95	4	.05	.85	4	.05	.85	4	.15	1.05	4	.05	.7	4	.00

TABLE 5 (*continued*)

Hot

Subject	*G.*			*Co.*			*K.*			*H.*			*Cl.*		
Region	*Med.*	*No. of Cases*	*P.E.*	*Med.*	*No. of Cases*	*P.E.*	*Med.*	*No. of Cases*	*P.E.*	*Med.*	*No. of Cases*	*P.E.*	*Med.*	*No. of Cases*	*P.E.*
Right arm	1.0	12	.05	1.1	8	.10	1.0	12	.15	1.2	12	.10	.8	12	.00
Left arm	.9	12	.10	1.0	12	.10	1.05	12	.10	1.5	11	.45	.8	12	.00
Head	.95	8	.10	1.1	8	.10	.95	8	.05	1.2	8	.10	.8	8	.00
Right leg	1.0	6	.05	1.3	6	.10	1.15	6	.10	1.7	6	.10	1.0	6	.05
Left leg	1.0	6	.10	1.35	6	.10	1.35	6	.25	1.4	6	.20	.95	6	.15
Abdomen	.95	4	.05	1.0	4	.05	1.0	3	.00	1.2	4	.00	.8	4	.00
Back	1.0	3	.00	1.1	4	.00	1.1	3	.20	1.25	4	.05	.9	3	.00

TABLE 6

MEDIAN REACTION-TIME FOR SINGLE STIMULI—GROUPED BY REGIONS

	Cold			*Hot*		
	Med.	*No. of Cases*	*P.E.*	*Med.*	*No. of Cases*	*P.E.*
Hand—dorsal (Areas 3, 4)	1.0	19	.05	1.1	20	.15
Hand—ventral (Areas 11, 12)	1.0	20	.15	.95	20	.15
Arm—dorsal (Areas 5, 6, 7, 8)	1.0	35	.15	1.0	40	.10
Arm—ventral (Areas 15, 16, 17, 18)	1.0	40	.10	1.0	39	.15
Face (Areas 19, 21, 22)	1.1	30	.10	1.0	30	.20
Neck—dorsal (Area 20)	1.1	10	.15	1.0	10	.20
Leg (Areas 23, 24)	1.15	16	.15	1.2	20	.20
Thigh (Areas 29, 30)	1.0	17	.15	1.05	20	.15
Abdomen (Areas 35, 36)	.9	19	.10	1.0	19	.10
Back (Areas 39, 40)	.9	20	.10	1.1	17	*.15

NOTE: The lack of a complete total for any region indicates that the remaining responses were "cold-hot" or "hot-cold."

* P.E. in these tables is really Q, the semi-interquartile range, or the distance above and below the median that will include fifty per cent. of the cases.

TABLE 7

MEDIAN REACTION-TIME OF H-C RESPONSES

Subject	*G.*			*Co.*			*K.*			*H.*			*Cl.*		
Region	*1st Int.*	*2nd Int.*	*No. of Cases*	*1st Int.*	*2nd Int.*	*No. of Cases*	*1st Int.*	*2nd Int.*	*No. of Cases*	*1st Int.*	*2nd Int.*	*No. of Cases*	*1st Int.*	*2nd Int.*	*No. of Cases*
Right arm	—	—	—	1.2	.4	19	1.2	.4	13	1.4	.95	12	.9	.4	11
Left arm	1.0	.35	6	1.1	.5	20	1.25	.4	12	1.6	1.0	9	.8	.4	13
Head	—	—	—	1.1	.45	6	.95	.4	4	1.4	.8	3	.8	.4	3
Right leg	—	—	—	—	—	—	1.2	.4	7	1.6	.9	3	—	—	—
Left leg	—	—	—	1.3	.75	7	1.4	.4	7	—	—	—	—	—	—
Abdomen	—	—	—	1.3	.5	6	—	—	—	1.6	.5	3	—	—	—
Back	.8	.4	3	1.1	.6	3	—	—	—	1.2	.5	3	—	—	—

TABLE 8

MEDIAN REACTION-TIME FOR C–H RESPONSES—GROUPED BY SUBJECTS

First Interval

Subject	*G.*			*Co.*			*K.*			*H.*			*Cl.*		
	No. of Cases	*Med.*	*P.E.*	*No. of Cases*	*Med.*	*P.E.*	*No. of Cases*	*Med.*	*P.E.*	*No. of Cases*	*Med.*	*P.E.*	*No. of Cases*	*Med.*	*P.E.*
Right arm	31	1.0	.05	13	1.1	.10	14	1.2	.20	12	1.3	.15	21	.8	.10
Left arm	29	1.0	.10	15	1.1	.10	15	1.1	.20	19	1.3	.10	18	.85	.10
Head	12	1.0	.10	10	1.1	.20	11	1.0	.15	12	1.4	.05	11	.9	.05
Right leg	21	1.0	.10	19	1.2	.20	13	1.1	.15	11	1.5	.20	22	.8	.05
Left leg	23	1.0	.10	12	1.3	.15	14	1.1	.10	17	1.4	.15	19	.9	.10
Abdomen	16	.9	.05	11	1.0	.15	14	1.0	.05	11	1.2	.15	15	.8	.05
Back	13	1.0	.15	14	1.0	.10	13	1.0	.10	15	1.2	.10	14	.8	.00

Second Interval

Subject	*G.*			*Co.*			*K.*			*H.*			*Cl.*		
	No. of Cases	*Med.*	*P.E.*	*No. of Cases*	*Med.*	*P.E.*	*No. of Cases*	*Med.*	*P.E.*	*No. of Cases*	*Med.*	*P.E.*	*No. of Cases*	*Med.*	*P.E.*
Right arm	31	.4	.00	13	.4	.00	14	.4	.10	12	.65	.40	21	.4	.05
Left arm	29	.4	.05	15	.4	.00	15	.4	.00	19	.7	.20	18	.4	.05
Head	12	.4	.10	10	.45	.05	11	.4	.05	12	.7	.10	11	.5	.10
Right leg	21	.4	.10	19	.6	.20	13	.4	.05	11	.6	.20	22	.8	.30
Left leg	23	.4	.05	12	.4	.00	14	.55	.15	17	.6	.20	19	1.0	.45
Abdomen	16	.4	.00	11	.4	.10	14	.4	.05	11	.5	.10	15	.4	.00
Back	13	.4	.05	14	.45	.10	13	.4	.00	15	.5	.10	14	.5	.10

TABLE 9

MEDIAN REACTION-TIME FOR C–H RESPONSES—GROUPED BY REGIONS

Region	*No. of Cases*	*First Interval*		*Second Interval*	
		Med.	*P.E.*	*Med.*	*P.E.*
Hand—dorsal (Areas 1–4)	50	1.1	.15	.4	.10
Hand—ventral (Areas 11–14)	41	1.1	.15	.4	.05
Arm—dorsal (Areas 5–8)	50	1.0	.15	.4	.05
Arm—ventral (Areas 15–18)	46	.95	.18	.4	.00
Face (Areas 19, 21, 22)	40	1.0	.10	.4	.10
Neck—dorsal (Area 20)	16	1.15	.20	.5	.10
Leg (Areas 23–26)	53	1.1	.25	.6	.20
Thigh (Areas 27–30)	71	1.0	.20	.4	.05
Foot (Areas 31–32.1)	49	1.1	.20	.8	.35
Abdomen (Areas 33–36)	67	1.0	.10	.4	.05
Back (Areas 37–40)	69	1.0	.15	.4	.05

RESULTS

With these precautions concerning the reliability of the method we may consider the results. Table 1 presents the individual responses in detail to double stimulations for the forty areas. Table 2 presents the detailed responses to single stimulations.

The first thing that may be noticed in a rapid inspection of Table 1 is the considerable number of responses that were given in the order *hot-cold*. Table 3 shows the proportion of responses in each order for each individual.

TABLE 10

COMPARISON OF REACTION TO HOT IN SINGLE AND DOUBLE STIMULI

	Double Stimulus C–H Resp. (i. e., Total Time)		*Single Stimulus*	*Difference*
	Med.	*P.E.*	*Med.*	
Hand—dorsal	1.5	.25	1.1	.4
Hand—ventral	1.5	.25	.95	.55
Arm—dorsal	1.5	.15	1.0	.5
Arm—ventral	1.3	.15	1.0	.3
Face	1.5	.15	1.0	.5
Neck—dorsal	1.7	.25	1.0	.7
Leg	2.0	.25	1.2	.8
Thigh	1.4	.15	1.05	.35
Foot	2.0	.30	1.2	.8
Abdomen	1.4	.20	1.0	.4
Back	1.4	.15	1.1	.3

The averages show that on the arms the percentage of C–H responses is only slightly greater than half—53.6 per cent. and 54.8 per cent., while on the abdomen and back it rises to 82.2 per cent. and 82.8 per cent. respectively. These relations do not hold exactly among individuals, but a tendency toward greater constancy for the latter regions is shown by the fact that on these regions, in only two cases does the per cent. fall below 80, while on the arms (with the exception of Subject G., whose per cents. are all high), no case rises above 57 per cent. As regards the H–C responses, the highest percentages fall on the arms, on the average, and for all subjects except G.; but the percentages on abdomen and back are not significantly smaller than on legs. Individuals vary greatly—from G., who has no higher than 19 per cent. of H–C responses for any region, and no less than 81 per cent. of C–H responses, to Co., who has as high as 59 per cent. of H–C responses for one region and only 34 per cent. of C–H responses for another.

With these responses may be compared the cases in which only one response was made to the double stimulus (Table 4). It will be noticed from Table 1 that one subject, K, sometimes reported that both sensations were perceived simultaneously. This fact will be considered later. Excluding these responses of K., we find that there are 71 instances (out of about 800 responses) when only one of the two stimuli is reported (*v.* Table 4). Of these, 40 are cold and 31 hot—a nearly equal distribution, but this is produced by the responses of one individual, H., who gave 23 hot responses; otherwise, the balance is in favor of the cold. With the exception of H.'s responses (which will be taken up again), nothing of particular significance appears from this table. These responses are probably to be explained by lapses of attention or slight inequalities in the stimulus.

The facts of Table 2 are more easily understood from Tables 5 and 6, and Figures 1 and 2, which attempt to present graphically the distribution of the reaction-times to separate cold and hot stimuli. The most striking thing about the distribution is the amount of overlapping in the reaction-times of cold and hot. The range in both cases is about the same, with a slightly greater variability for hot. The medians show no consistent difference for the two sorts of stimuli. In 20 cases out of 35 (*cf.* Table 5), the median cold reaction is shorter. In 14 of these, it is shorter by as much as .1 second, and in 9, by .2 second or more. In 10 cases the median for hot is shorter, and in the remaining 5 cases the medians are the same. This may be due to the rather rough method of measurement, since other observers have found a clearer difference; on the other hand, if the measurement adequately represents the fact, it explains, perhaps, the rather large number of responses to double stimuli that occurred in the order, *hot-cold.* The averages for the individual areas (*cf.* Table 2) show the reaction-time for hot to be greater than for cold in 17 out of 26 cases—a slightly greater percentage of cases than appears from the medians of the grouped areas (Table 6), although in only 8 of these instances was the difference in time as much as .1 second.

It is of interest in passing to compare the median reaction-time to cold and hot separately (Table 5) with the percentages of C–H and H–C responses (Table 3), though it must be remembered that the medians of Table 5 are computed on too few cases to be very reliable. Still it is rather curious that Subject G., who showed fewest cases of H–C responses, nevertheless has more areas that give a shorter median reaction to hot than any other subject—three cases out of seven. A certain tendency toward consistency is, however, suggested by a comparison of Tables 3 and 6. Table 3 shows the greatest number of H–C responses, as well as the least number of C–H responses to occur on the arms. Table 6 shows that, with the exception of the back of the hand, the other areas on the arms have less than .1 second difference in the medians for cold and hot, one being greater for hot than for cold, one less, and one equal to cold. The abdomen and back, on the other hand, show .1 and .2 second difference respectively, the median for cold being shorter; and these areas likewise have the greatest number of C–H responses. This seems to indicate that the distribution of C–H and H–C responses is a real one and is not due exclusively to faults or inaccuracies in the procedure.

One other thing may be noticed about Table 6—the instances of double responses to the single stimulus. Some regions show an incomplete total for the group of areas designated, and the remaining number of responses were either "cold-hot" or "hot-cold." Fifteen such responses were to cold, five to hot—that is, cold was more often felt as cold-hot than was hot felt as hot-cold. (H–C responses to cold and C–H responses to hot did not occur.) This is probably another illustration of the fact, often mentioned, that hot is a more difficult, less certain sensation than cold; so that any change or imagined change after cold is apt to be reported as hot. Subject Cl. so explained to herself several cases in which she reported the cold stimulus as "cold-hot" and then immediately corrected herself. (These cases, if included in the count, would further increase the difference.) Possibly the slight change in sensation was due to inexactness in setting down the blocks simultaneously and with equal pressure. The greater number of cold stimuli perceived as cold-hot may also, perhaps, be in part the effect of suggestion—more responses to double stimuli occurred with cold first; therefore greater likelihood existed that hot would follow cold than *vice versa.*

When we turn to the double responses, the hot-cold responses to double stimuli are not numerous enough to form a very accurate basis of comparison with the cold-hot responses. Table 7 shows the medians for those regions that have as many as three such responses. Of course most of these have a high degree of unreliability, but no particular differences from the distribution of cold-hot responses appear.

These responses may also be compared with the C–H responses in regard to the length of the interval between the first and second responses. In order so to compare them, the ratio of the second response to the first was determined for each response. The distribution of these ratios is presented in Figure 3. The medians for both sorts of responses are indicated by circles and crosses respectively. In twenty-two cases there were at least three H–C responses for a given region. Of these, five are equal to the median ratio for C–H responses for that region, five are less, and twelve are greater. That is, when the response occurred in the order *hot-cold*, there was a slight tendency for the second interval to be longer in relation to the first than when the response occurred in the order *cold-hot.* This is rather unexpected, and may perhaps be considered an intimation that in such cases the blocks were not applied quite simultaneously.

We may turn now to a study of the cases in which the response was given in the order

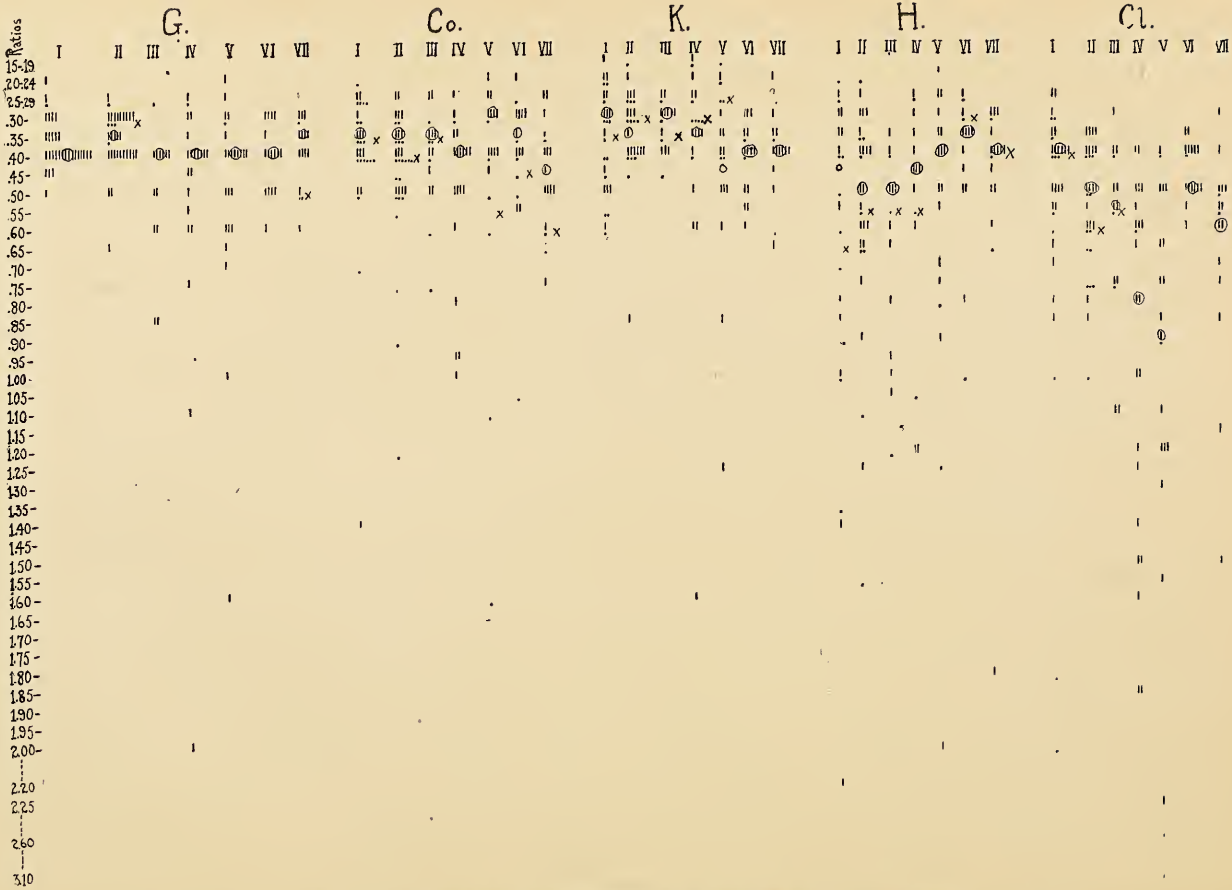
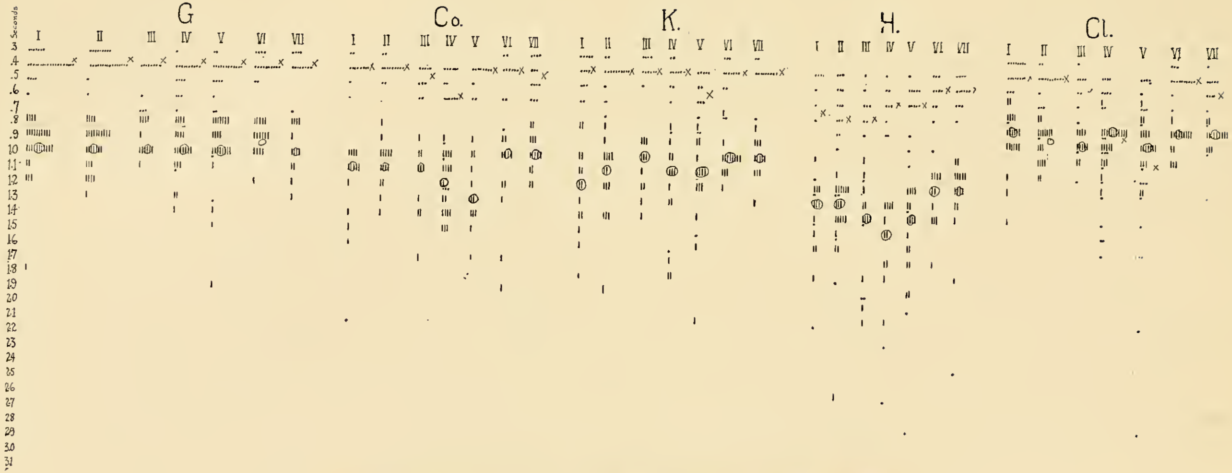


FIG. 3. DISTRIBUTION OF RATIOS $\left(\frac{\text{2nd Interval}}{\text{1st Interval}}\right)$ OF COLD–HOT AND HOT–COLD RESPONSES

Strokes indicate ratios for C–H, dots for H–C responses; circles and crosses show the median ratios for C–H and H–C responses respectively for each region, having at least three responses of the given sort



[Fig. 4. Distribution of Cold–Hot Responses Grouped According to Subjects

Strokes indicate length of first interval; dots length of second *cold-hot* (*v.* Tables 8 and 9, Figures 4 and 5). Figure 4 shows the distribution for each individual and brings out such individual differences as there are. Subjects G., Co., and K. are quite similar as to median times and distribution; H. has somewhat longer times and more extreme reactions, but a similar difference in the median time of the two intervals; Cl. has a curious distribution, in that the median time for the second interval is relatively long—longer even than for the first interval, in one case. Four subjects show less variability for the second interval than for the first; that is, the second interval follows the first at a fairly constant interval of about .4 second, whatever the length of the first interval. In the case of Cl., however, the reverse is true. This accords with the fact shown by the distribution of ratios (*cf.* Figure 3): the ratio of the second interval to the first varies more than the second interval. Individuals vary least in reaction-time on abdomen and back (range of medians for these regions, .8–1.2 seconds—*cf.* Table 8), and most on legs (range of medians, .8–1.5 seconds).

The double stimulus, when cold is perceived first, does not appear much to affect the reaction-time to cold. Comparing the reaction-time to cold in the case of single and double stimulations (*cf.* Table 6 and Table 9), we find that three are shorter for the double than for the single stimulus, five are longer, and three are equal in the two cases.

The effect on the reaction-time to hot is, naturally, to increase it. This may be seen by comparing the medians for the total reaction-time of C–H responses (*i. e.*, the time for the perception of hot in the double stimulations) with the medians for the reaction to hot separately. Table 10 presents the comparison.

The difference is about .4 second, on the average, but it is noteworthy that on the foot, leg and neck, the time is considerably increased for the double stimulus. This increase is due partly to a delay in the perception of hot for those regions, as the table shows. But this is not enough to explain the amount of difference in the reaction of these regions to double stimulations; the greater delay in this case must be in some way dependent on the double stimulation. It is possible that this is occasioned by the phenomenon referred to by Alrutz and Goldscheider as the *Anschwellung* of the sensation—the increase in intensity of the sensation immediately after the first perception of it—which is certainly most noticeable on the feet and legs. At this stage of the experiment, however, nothing definite can be said on this point.

Since the purpose of this paper is chiefly to present the facts as they appeared from an examination of normal subjects, the paper will close with a summary of the individual cases. The five individuals chosen were presumably normal. They were not given a neurological examination, but they were all personally known to the experimenter.

TABLE 11

SUMMARY OF INDIVIDUAL DIFFERENCES

	Total No. of Resp. to Double Stim.			*Reactions Giving Response C—H*					*Ratio of 2nd Intv. to 1st*			*Reaction to Sing. Stim.*
				Reactions Greater than 1.4 Sec.	*Range of Medians of Different Regions*		*Areas Having less than 2 Resp.*	*Total Time Greater than 2.5 Sec.*	*Single Ratios*		*Medians*	*Med. for H. less than C*
									<.25	*>1.0*	*>.44*	
	C—H	*H—C*	*Sing.*	*No. of Cases*	*1st Intv.*	*2nd Intv.*	*No. of Cases*	*No. of Cases*	*No. of Cases*			*No. of Cases*
G.	144	13	5	4	.1 sec.	0 sec.	0	0	2	3	0	3
Co.	94	68	15	11	.3	.2	10	2	3	5	1	2
K.	92	47	5*	11	.2	.2	10	3	14	2	1	2
H.	97	34	35	37	.3	.25	9	17	4	15	6	1
Cl.	120	39	10	7	.1	.6	3	4	0	23	6	2

* Thirteen cases were reported as simultaneous.

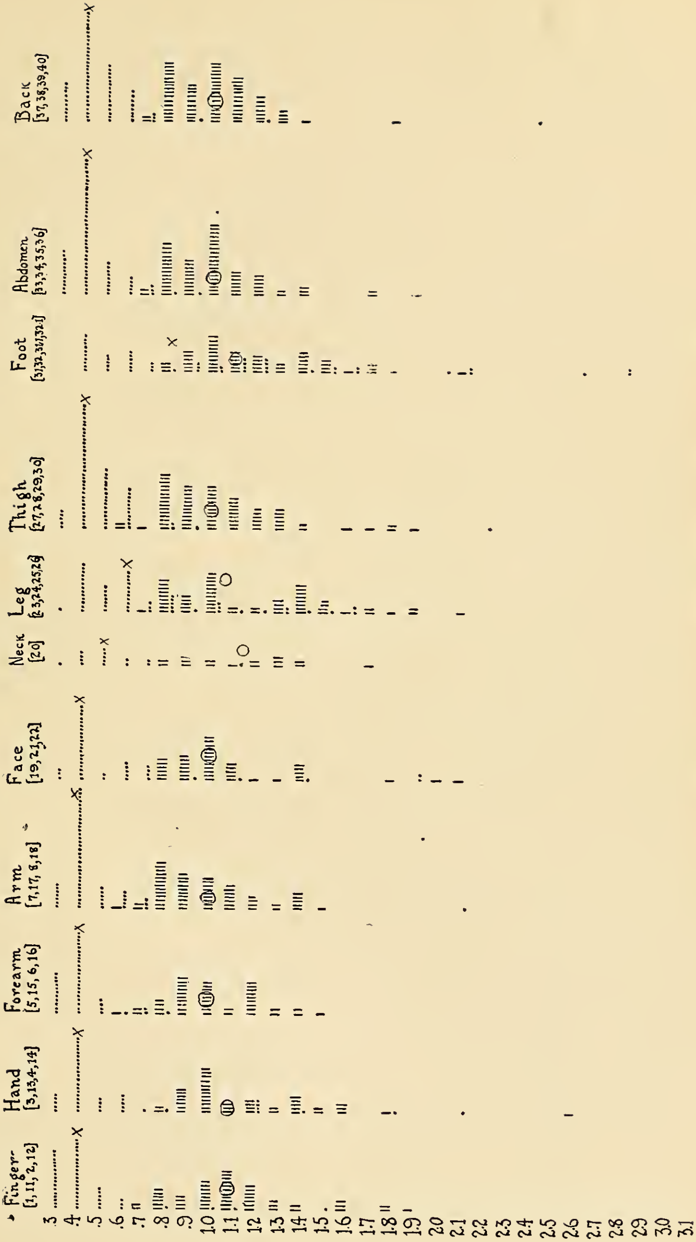


Fig. 5. Distribution of Cold–Hot Responses Grouped According to Regions

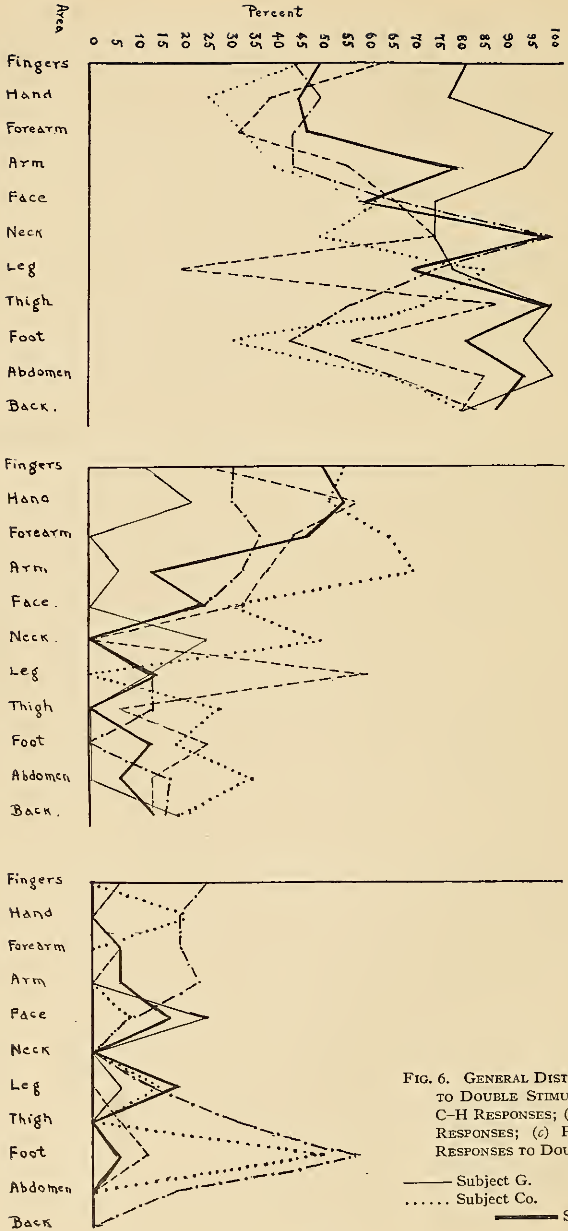


FIG. 6. GENERAL DISTRIBUTION OF RESPONSES TO DOUBLE STIMULI; (*a*) PERCENTAGE OF C–H RESPONSES; (*b*) PERCENTAGE OF H–C RESPONSES; (*c*) PERCENTAGE OF SINGLE RESPONSES TO DOUBLE STIMULI

——— Subject G.
...... Subject Co.
- - - - Subject K.
–.–. Subject H.
▬▬▬ Subject Cl.

Subject G.—A young woman of about twenty-one years of age, a student of domestic science, without any known nervous or temperamental abnormalities.

Compared with other subjects, G. showed a greater constancy of reaction in every respect. All the figures of distribution show a close grouping of her reaction-times—only four instances in the C–H responses when the reaction was greater than 1.4 seconds, only eighteen cases when the hot reaction preceded the cold, five reactions in which only one of the two stimuli was perceived, scarcely any variation in the median time for different areas for either first or second interval—but, on the other hand, three areas where the median for the separate hot stimulus was shorter than the median for cold; a close grouping of ratios, and no individual area which did not give at least two reactions in the order *cold-hot*. On the whole, G. is rather an ideal illustration of the norm.

Subject Co.—A woman of about fifty years of age, without any known abnormalities.

The distribution of Co.'s responses is more variable but shows the same general characteristics as G.'s, except in the number of H–C responses. Seventy cases occurred, most of them on the arm, hand, or foot. It is to be noted (*cf.* Table 1) that the H–C responses that were repeated gave the same kind of response only on arm, hand, and foot—never on other regions. It is also noticeable that Area 11 gave no C–H responses but six in the order H–C; Area 14, no C–H responses but five H–C; Areas 3, 31, and 32.1 gave three single responses, and in the case of Area 31, all three were "cold." Co. shows a somewhat greater variability for different regions of the body than does G., with shortest median times on abdomen and back.

Subject K.—A young woman of about thirty years of age, a graduate student in psychology—no abnormalities known.

K. also shows the same general form of distribution, with an intermediate number of forty-seven H–C responses. K. however, reported thirteen times that the sensation was simultaneous. This is rather curious, since it occurred in no other subject, and it was probably the effect of suggestion. K. had heard a little about the experiment before acting as subject, and while she did not know the procedure, she evidently speculated a little about it. Toward the end of the second day she remarked that she supposed the blocks were put down simultaneously and she felt one before the other, because she did not perceive two contacts! It will be noticed from Table 1 that on the first day no cases of this sort appear. Nevertheless K. appeared to have some hesitancy in determining which sensation came first, and it is probably in consequence of this that she has fourteen ratios (compared with a small number in other subjects) less than .25, which indicates a relatively long first interval.

Subject H.—A woman of about forty-five years of age, janitress of a flat, generally regarded as of a rather nervous temperament.

H. deviates somewhat from the general distribution of the first three subjects. Her reaction-time is longer, and she has more extreme reactions—thirty-seven of her C–H responses are greater than 1.4 seconds. Her total times are in seventeen instances greater than 2.5 seconds (compared with eight, the maximum for any other subject). Her ratios are also longer, fifteen being greater than 1.0, and six medians being more than .40–.44, the most frequent ratio. H. has thirty-five cases of a single response to a double stimulus—three areas giving a single response as many as three times out of four. Twenty-three of these were hot and twelve cold, reversing the proportions of every other subject but K., in whom the two sorts were practically equal. In contrast to this, it will be noticed from Table 1, that not infrequently H. responded by "warm" instead of "hot," although she was not asked to differentiate. It is true that for this subject on the first day the temperature of the hot stimulus fell to 38°; however, most of the "warm" responses did not occur on this day, and on other days the temperature was not below 40°.

Subject Cl.—A young woman of about thirty years of age, a graduate student in psychology. On the second day Cl. was recovering

TABLE 12

Summary of Reactions

	Cold—Separate		*Hot—Separate*		*Cold—Hot*				*Hot—Cold*			
					1st Interval		*2nd Interval*		*1st Interval*		*2nd Interval*	
	I	*II*	*III*	*IV*	*V*	*VI*	*VII*	*VIII*	*IX*	*X*	*XI*	*XII*
Fingers	10	8–11	9	9–12	10	10–12	4	3– 5	12	10–13	5	4– 7
Hand	10	10–12	11	9–12	10	10–14	4	4– 6	12	10–14	4	4– 6
Forearm	10	9–12	10	9–12	10	9–12	4	3– 4	11	9–14	5	4– 6
Arm	10	8–11	10	8–11	10	8–11	4	4– 5	12	10–14	5	4– 7
Face	11	10–12	10	8–12	10	9–11	4	4– 6	11	10–14	5	4– 6
Neck—dorsal	11	9–12	10	8–12	10	9–11	4	4– 6	14	10–16*	4	4–12*
Leg	11	10–13	12	10–14	11	9–14	6	4– 8	12	10–16	4	4
Thigh	10	9–12	10	10–12	10	8–11	4	4– 5	12	12–15	5	4– 9
Foot	12	10–13	12	11–15	11	10–14	8	5–12	16	15–16	8	5–14
Abdomen	9	8–10	10	9–11	10	9–11	4	4– 5	13	10–16	5	4– 6
Back	9	8–10	11	9–12	10	8–11	4	4– 6	10	9–11	5	4– 6

* Total range: group included only three cases.

from an attack of neuritis in her left arm. This does not seem to have changed the character of her responses for this day or for her left arm in general.

The general character of Cl.'s responses shows quite individual characteristics — a markedly short reaction-time to hot and cold separately and to cold in the case of the double stimuli, but a delay in the reaction to hot on the legs, which made the median for hot, in the C–H responses, as long or longer than the median for cold. She has few extreme reactions, but all of them are to hot stimuli. Of the ten instances of single reaction to double stimuli, only two were "hot." In Figure 2 extreme reactions to hot separately may be noted on the left arm and right leg, while in Figure 4 nearly all the reactions that are extreme occur on the right and left legs and not at all on the left arm. Her ratios tend to be longer than the others; six medians are over .40–.44, and twenty-three separate ratios are greater than 1.0.

CONCLUSIONS

In so far as these five subjects may be representative of the normal reaction to cold and hot stimulation under the conditions of this experiment, we may summarize the results as follows:

1. More than half of the total number of reactions to simultaneous cold and hot stimulation elicit first cold, then hot. The back and abdomen have a higher percentage than other regions of such responses.

2. The proportion of responses to double stimulation that does not include both responses is small—8 per cent. on the average, with a minimum of 3 per cent. and a maximum of 20 per cent.

3. The reaction-time to cold and hot stimuli separately does not show any distinct difference, as measured in this way, except perhaps on the abdomen and back.

4. The length of the first interval, when "cold" is the first response to the double stimulation, is about 1.0 second; the length of the second interval, about .4 second, on the average. A second interval of more than 1.0 second is rare.

5. The order in which the response occurs —whether "cold-hot" or "hot-cold"—is probably dependent in part on the simultaneity of application and in part on physiological conditions.

6. Individual differences in all of these respects are so considerable that a wide deviation from the average must be admitted as normal.

7. The method, though not accurate within very fine limits, gives a clinical picture sufficiently precise to show individual differences among normal subjects, and should

TABLE 12

SUMMARY OF REACTIONS

	% C—H	*% H—C*	*% Sing.*	*% ?*
	XIII	*XIV*	*XV*	*XVI*
Fingers	55.6	34.6	8.8	1.1
Hand	47.1	41.5	7.8	3.3
Forearm	50.6	36.1	6.0	3.6
Arm	58.2	32.6	7.4	1.3
Face	65.5	23.1	11.4	—
Neck—dorsal	84.2	15.8	—	—
Leg	64.6	18.3	9.7	7.2
Thigh	83.1	9.6	7.2	—
Foot	61.2	11.2	25.0	2.4
Abdomen	81.7	14.6	3.7	—
Back	83.1	15.7	—	1.2

therefore be useful in dealing with abnormal cases.

For convenience of reference, a table summarizing the data is appended. The first two columns of the table give, respectively, the median reaction-time in tenths of a second, and the limits between which 50 per cent. of the cases are included, for responses to separate *cold* stimuli. Columns III and IV give the same facts for separate *hot* stimuli. Columns V–XII contain similar data for responses to *double* stimuli, first for Cold-Hot, then for Hot-Cold reactions, and for each interval. Columns XIII–XVI show the proportion of double stimuli that gave responses of each kind: C–H, H–C, Cold or Hot alone, and Doubtful (where the priority of either could not be decided).

If individuals are to be compared with this table, it must be remembered that a subject's median reaction-time may lie anywhere within the limits of the probable error or even slightly outside of these limits and still be normal. The percentage of stimuli giving any particular sort of response is even more subject to individual variation. All that it would be safe to say about the normal reaction in this respect is that a majority of the total responses of an individual should be Cold-Hot, though on any region except the face, thigh, abdomen, and back the percentage may fall below fifty; and further, that a very small proportion of the total will fail to give both responses, and that the neck, back, and abdomen are least likely to present this type. The graphs in Figure 6 will make this clear.

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HEMORRHAGE INTO THE BASAL CISTERNS AND VENTRICLES OF THE BRAIN—A CRITICAL ANALYSIS OF THE SYNDROME PRODUCED BY THIS LESION*

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In its general features the syndrome consequent upon hemorrhage into the basal cisterns and ventricles of the brain is well known. Many of its more intimate details, however, still present opportunities for further study and it is in the hope of contributing to knowledge in these respects that the following case is reported. We desire to point out the correlation between the pathological course of events and the symptoms as we interpret them. The case has additional interest because of the unusual manner in which the basal cisterns and the ventricles appear to have been successively flooded.

HISTORY.—The patient, a woman, aged forty-eight years, married and the mother of five healthy children, was suddenly stricken with intense pain in the head, rapidly became comatose and died in thirteen hours.

Previous History.—Her career, both in early life and after her marriage, was notable for its intellectual activity. She was unusually talented, being an authoress, translator, and writer upon scientific subjects. Her previous history shows that she had suffered from no serious illness, nor was she subject to fainting spells or unusually severe headaches. In her forty-first year she was operated upon for the repair of cervical laceration from which she made a complete recovery with the relief of symptoms. She had one miscarriage at about three months in her forty-fourth year, following over-exertion, while the pregnancy prior to this was an ectopic gestation, from which she made an uneventful recovery after her condition had been *in extremis*. In her habits she followed moderation in all things.

Family History.—The family history affords some evidence of early cardiovascular degeneration with a distinct tendency to apoplexy. There was no epilepsy, no insanity, and no hereditary nervous diseases among her relatives.

Prodromal Period.—There are certain features concerning her condition and activity during the several weeks prior to the onset of her disease which seem to be worthy of note. In so far as it is possible to determine, there was a period of perhaps a month in which certain slight changes in her appearance and attitude gradually became cumulative until her condition seems to have assumed, in the light of later developments, a definite prodromal phase. At first these changes were not particularly evident in her appearance although she gave the impression at times of great fatigue in her facial expression and her color was frequently higher than was normal for her. It was in a seemingly increased pressure of activity, however, that the most notable alteration was manifest. This, in its inception, was scarcely more than might have been expected of a usually busy woman. Shortly, an element of unrest and even restlessness became apparent. She devoted herself to her domestic duties, her social relations and her literary activities with an interest almost excessive even for a woman of her temperament. She became easily fatigued from these pursuits and her intimates, especially in the family, noticed an irritability which was foreign to her usually serene nature.

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* Presented at the Clinical Conference, March 11, 1918

Her sleep was considerably disturbed. This may have been due to slight digestive disorders which made their appearance about this time, or what is more likely, to an irregular, fitful headache of which she began to complain quite persistently in the two weeks preceding her death. It was this headache probably which led to an increasing inability of concentrating her attention on any definite interest for more than a short time. The headache, although not constant, gained in severity and became a very frequent complaint in her last two weeks.

The Onset.—After a busy day, March 2, 1916, she retired at midnight and was still awake when called by her daughter who asked her to come upstairs. The patient started immediately to comply and ran hurriedly up the stairs. Almost at once she felt a severe, sharp pain through her head. It seemed at first to stun her. At once she threw herself on a bed but soon sat up, held her head and began to cry with pain. She was conscious and able to give a strikingly graphic description of the pain which she said came as increasing waves, each one of which seemed to her as though about to burst open her head. She recognized the danger of her condition saying that she must be suffering from a hemorrhage into her brain because her feelings were so similar to those experienced at the time of her hemorrhage due to the ectopic gestation a few years before. The headache of which she complained was at first located in the top of her head but as this grew in intensity she spoke of a spread of the pain to the back of her head, then to her neck, from which point it rapidly extended to a level between the shoulders, down the spine into the hips and finally into the rectum. During the entire time the pain in the head was steadily increasing in severity and becoming continuous. Her speech grew less coherent, her face became suffused and cyanotic and finally, as if losing all control of her emotive expressions under the influence of her intolerable suffering, she screamed loudly and fell unconscious. The duration of this period was five or ten minutes. As her head fell upon the pillow, the muscles of the neck contracted so that her face was turned to the right and simultaneously both eyes were drawn over to that direction. Shortly after she became unconscious, she had a severe attack of projectile vomiting followed by retching and also involuntary defecation with forceful expulsion of much flatus.

The first neurological examination was made one hour after the onset, the findings of which are as follows:

INITIAL NEUROLOGICAL STATUS

APPEARANCE AND ATTITUDE.—The patient was found recumbent in a supine position. The head was drawn so that the chin pointed to the right. The eyes were also drawn to the right; the eyelids were closed. Both arms were held in a moderate degree of abduction from the chest, the forearms slightly flexed at the elbows, the wrists extended, with the fingers extended at the proximal and flexed at the distal phalangeal joints. The face was cyanotic as were also the extremities.

VOLUNTARY MOVEMENTS.—The arms, held rigid in the posture above described, were moved slowly and rhythmically in abduction and adduction. In these contractions the successive abductions and adductions were both forceful, and although the adductions were sufficient to dominate the movements, they were rigorously opposed by the abductions. Having been adducted, the next succeeding movement was an abduction which appeared to be due rather to a relaxation of the adductors than to any accession of muscular contraction in the abductors. This series of alternate abductions and adductions was carried on at the rate of about eighteen to twenty per minute, the adductor movement in the majority of instances being synchronous with inspiration. The amplitude of contractions never carried the arm into actual contact with the thorax, nor did the abduction ever exceed an excursion of ten degrees. Each successive contraction, while corresponding to the others in general characteristics, did not necessarily produce the same range of motion, although each contraction had the same tense rigidity and never, at any time, showed a tendency to develop what might correctly be called a clonic element. The trunk musculature did not participate in these contractions.

In a remarkably similar manner the lower extremities took part in the convulsive movements. The thighs, slightly flexed upon the pelvis, were drawn into adduction and still more marked flexion at the hips. Succeeding this movement, the thighs were abducted and more nearly extended. While these spasmodic movements were going on in the thighs, the legs were held in semiflexion at the knee, and the feet in extension. The movements of the lower extremities were synchronous with those of the arms.

The convulsive movements observed at this initial period had the following characteristics:

1. A marked hypertonus in the upper and lower extremities.
2. Rhythmical contractions of the axio-appendicular musculature, producing alternate abduction and adduction of the arms and thighs. These movements were synchronous with respiration.
3. Forceful tonic contractions of the appendicular musculature, producing fixed attitudes of the forearms, hands, and fingers, legs and feet.

About twenty minutes after the onset she had a violent projectile emesis followed by considerable retching and, after a short interval of rest, a second attack of similar character. Simultaneous with the vomiting, forceful defecation occurred accompanied by the expulsion of much flatus. Defecation of the same general nature continued off and on throughout the next hour. The abdomen was slightly distended but not tympanitic. Almost immediately after the onset of the coma and more particularly after the violent emesis the respiratory activity assumed unfavorable characteristics. While not entirely of the Cheyne-Stokes type, there was a distinct tendency to diminution of respiratory excursion; sometimes in the typical series, but more often in an irregular manner. The respiration was now slow, now rapid, but at all times arhythmical, with the interpolation of many deep sighing inspirations. The feature of the entire respiratory movement during the early period of the disease was its marked irregularity in rate, rhythm, and excursion, indicating to one's mind a distinct imbalance in the central respiratory control. The breathing was accompanied by a slight stertor, also by some dilatation of the alæ nasæ and opening of the mouth synchronous with inspiration. The pulse was irregular in its rhythm with frequent dropped systole and marked fluctuation in tension. The pulse rate was eighty per minute and the blood pressure, taken at the brachial artery, showed systole at 220, mm. Hg.; diastole 100, mm. Hg.; pulse pressure 120. Not every systole was carried through at the same elevation and in consequence of this there was an unevenness in the force of the heart beat. These cardiac disturbances we interpreted as an imbalance in the sympathetic and autonomic control of cardiac activity. The heart sounds were normal except for a marked accentuation of the second aortic.

Coördination.—Such movements as resulted from the rhythmical contractions of the muscles in the arms and legs were well coördinated and, in their persistent repetition, followed the same excursion and had about the same amplitude. Other than the convulsive movements already mentioned, there were no further irregular, involuntary acts observed. The patient, at no time, made an effort to rise in bed, turn her head or otherwise alter the position which her body assumed when she fell in coma.

Reflexes.—An examination of the reflex status taken at this time showed the following conditions:

Reflexes	*Right*	*Left*	
Jaw	Active	Active	*Equal on both sides*
Biceps	Active	Active	
Olecranon	Active	Active	
Radial	Active	Active	
Wrist	Active	Active	
Knee	Very active	Very active	
Ciliospinal	Absent	Absent	
Epigastric	Active	Active	
Abdominal	Active	Active	
Babinski	Absent	Absent	
Chaddock	Absent	Absent	
Oppenheim	Absent	Absent	
Schäfer	Absent	Absent	
Gordon	Absent	Absent	
Periosteal	Absent	Absent	
Crossed Periosteal	Absent	Absent	
Marie-Foix	Absent	Absent	
Clonus Jaw Wrist Knee-foot	Absent	Absent	

Skilled Movements.—Skilled movements and voluntary movements of all kinds were abolished immediately after the onset of the coma. There was, however, no evidence of paralysis and the constant rhythmical movements of the arms and legs gave evidence that the corticospinal transmission apparatus was still intact. All of the muscles of the body were in a state of hypertonus, but the degree of this muscular tension varied in different parts of the body. It was most marked in the upper and lower extremities and least pronounced in the trunk. A moderate degree of hypertonus was observed in the neck muscles, particularly those of the left side, in which the sternocleidomastoid was in active tonic contraction sufficient to rotate the head in such a way that the chin pointed toward the right. There was no actual stiff-neck nor was the hypertonus in any part so extreme as to give much resistance on passive movement. The increased muscular tone, however, did produce fixed attitudes of the fingers, hands, forearms and arms; feet, legs and thighs, as already described. No tremors were evident in any part of the facial musculature, in the tongue, hands or feet.

NERVE STATUS.—No abnormality was observed in this respect.

GENERAL SENSORY.—The general sensory responses were so poor as to leave them in doubt. Hyperextension at the wrist and elbow, as well as at the knee, seemed to cause a suspension of respiration and produce a slight corrugation of the brow. These stimuli were not met with any increased resistance in the parts tested. Pinching of the skin elicited no perceptible response. Somatic sensibility appeared to be almost, if not entirely, abolished in all qualities of sensation.

CRANIAL NERVES.—The cranial nerves of special sense, the olfactory, optic, and auditory, gave no evidences of sense perception. The eye grounds showed indistinct optic discs, elevated to the extent of one diopter on either side. The nerves innervating the ocular muscles showed no paretic defects. The eyes were held constantly in extreme right deviation.

There was no nystagmus and no ocular dissociation. The cephalogyric position of the head to the right corresponded with the oculogyric position of the eyes and this latter was interpreted as a manifestation of hypertonus in the ocular muscles. Both eyelids were closed. There was no blepharospasm. The eyes showed both conjunctival reflexes absent. Direct and consensual light reflexes were absent, right and left. The pupils were small and regular.

Facial expression was normal although at times there appeared to be a slight flattening of the right side of the face. There was no apparent defect in the muscles of mastication.

Deglutition was normal and the head held constantly turned to the right as described. It could, however, with a slight degree of force be turned to the left.

Speech and voice sounds were abolished. The tongue was held in the floor of the mouth, the latter slightly gaping.

Sphincteric control was lost in both bladder and rectum and there was an occasional forceful expulsion of urine and feces.

COURSE OF THE DISEASE

The entire duration of the disease from its inception until the death of the patient was thirteen hours. During this time a careful study was made in all the salient features of the patient's condition and the following record is given in detail in order that the clinical picture may be presented in its entirety.

Coma, which in the beginning was most profound, remained unaltered until the end. There were no periods in which somatesthetic perceptions were manifest with the possible exception of some slight resistive movements during the repeated readings of her blood pressure. These were not invariably observed at each reading and were more particularly noted in the sixth, seventh, and eighth hour of the disease. After this time even these movements were not in evidence. Some of the lower association reflexes, however, were retained, such as that of deglutition, for the patient took fluid when given to her by mouth and on several occasions coughed when the fluid irritated her throat. There was no apparent dissociation in the deglutition reflex and this continued so until the twelfth hour after which time no attempt was made to administer fluids by mouth.

The convulsive movements, noted at the onset of the disease, maintained their initial intensity for the first two hours but subsequently decreased both in force and excursion. They presented all of the characteristic features observed at the onset; namely, the rhythmical abduction and adduction contractions of the axio-appendicular muscles with tonic contraction of the appendicular musculature which tended to hold the extremities in a more or less marked degree of flexion.

During the fourth hour these convulsive movements attenuated and ceased nor did they recur except at very irregular intervals and for a few seconds at a time.

The projectile vomiting occurring at the outset was repeated but twice thereafter during the first two hours. No further emesis was observed. Defecation and urination continued involuntarily and the only difference apparent in these two functions was the less propulsive nature of defecation. Urine was obtained, a specimen of which showed nothing abnormal upon the routine analysis.

Respiratory Irregularities.—The respiratory activity manifested certain interesting changes the nature of which we shall attempt to explain in the discussion of all the symptoms. The outstanding feature of this alteration was the gradual respiratory compensation which appeared to take place in the first four hours. The respiration which, in the first hour after the onset showed a distinct tendency toward the Cheyne-Stokes type, retained this feature for nearly four hours. There were long periods in which the respiratory movements passed through a well-defined crescendo toward the end of which the excursion became slight and the inspiration shallow. Following this, there was a relatively long period of several seconds in which respiration was suspended. This was succeeded by deep and sighing inspirations accompanied by an adductor spasm in the arms and legs. The next few respiratory acts were regular in their rate and excursion and the inspiration in each instance was accompanied by the spasmodic movements above described. Expiration was accompanied by a pronounced stertor. In the fourth hour respiration became much more regular and remained in this condition until a few minutes before death. During this period the respiration averaged between 39 and 42 per minute. Throughout the entire course it was costo-abdominal in type.

Cardiac and Vascular Disturbances.—The circulatory status in the first two hours gave the appearance of a marked cyanosis in the face and the hands. The pulse at first was irregular in force and frequency and irregularly gave a dropped systole. This, like the respiratory irregularities, persisted through the first four hours, becoming less pronounced until finally in the fifth hour the pulse, although rapid, was steady, regular, and of good quality. This condition continued until the final stage of the disease when again a marked decompensation developed. During the first four hours the pulse rate ranged between 80 and 90 per minute. From the fifth hour until the end it ranged rather irregularly between 112 and 140.

The heart sounds were all well defined. There was a faint systolic murmur at the apex and a pronounced accentuation of the second aortic.

The blood pressure during the course of the disease showed a remarkably well-sustained systolic level of about 200 mm. of mercury for the first four hours. In the fifth, sixth, seventh, and eighth hours there was a slight depression in the systolic pressure at which level the pressure remained without much fluctuation. The diastolic pressure during this time held consistently to a level of 120 mm. and showed just as few variations as did the systolic line, the average pulse pressure during this period being between 75 and 80 mm. At the eighth hour the systolic pressure showed its first rise going suddenly to 230 mm. with no change whatsoever in the diastolic pressure. Hereafter, during the ninth hour, the systolic pressure slowly returned to 200 mm. in step-like fashion, while the diastolic pressure rose to 130 mm. where it remained. At the beginning of the tenth hour the systolic pressure again began to ascend, this time rather slowly, reaching 230 mm. at the end of seventy-five minutes, after which it returned to 220 mm. where it remained until just before the beginning of the thirteenth hour, when it suddenly went up to 270 mm. about ten minutes before the beginning of the terminal convulsion. Following this the descent was rapid, falling to 100 mm. in one hour just before death.

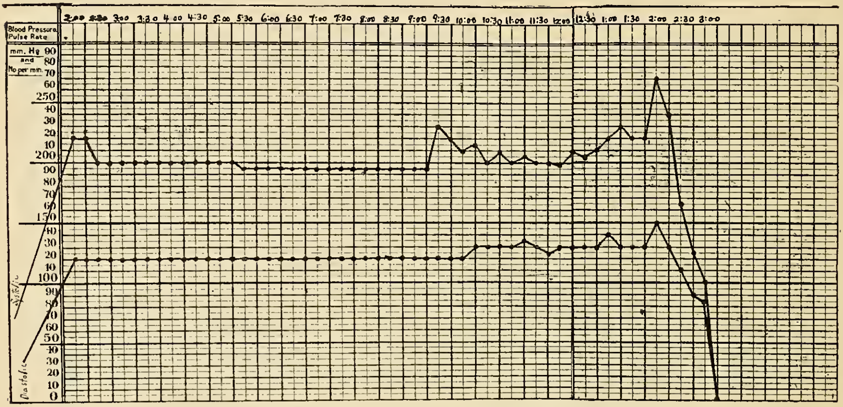

Chart Showing Variations in Diastolic and Systolic Blood Pressure

The diastolic pressure during this time maintained a surprisingly even level of about 120 mm. until the sudden rise of the systolic pressure when the diastolic itself ascended to 150 mm. and then fell with the systolic, but not as rapidly, reaching only 85 mm. just before death.

The pulse pressure in the last six hours, *i. e.*, up to the preterminal systolic rise, remained much as before, except that after the tenth hour it slowly rose with the increasing systolic pressure. When the systolic pressure reached 270 mm. the pulse pressure was 120 mm. Hereafter it rapidly decreased and at the last reading was 15 mm.

At the beginning of the second, third, and fourth hours respectively the patient received 1.120 gr. of aconitine by hypodermic. Again at the beginning of the eighth hour, hypodermatic treatment was resumed, the patient receiving gelseminine hydrochlorate (Merck) gr. 1/50 every thirty-five minutes for six doses.

Almost from the inception of the hemorrhage the temperature maintained a fairly even level at 101.° to 101.6° The initial rise immediately following the onset caused an elevation to 101.° where it remained with but two slight fluctuations until death. The principal features in the temperature curve are its sustained character and the absence of any antimortem rise.

Certain noteworthy changes occurred in the neurological status as determined by a second examination made nine hours after the onset. Most marked of these were the alterations in the deep reflexes. The tendon reflexes in the upper extremities manifested little if any change. The patellar and ankle reflexes were distinctly more active than at the first examination. The supra-umbilical and suprapubic, upper and lower lateral abdominals, although well marked at first, were now absent. A bilateral Babinski was also elicited at this time whereas earlier these reactions were not present. The Babinski was confirmed by a bilateral Chaddock. The Oppenheim, Schäfer, and Gordon reflexes were not observed. There was no periosteal reflex either direct or crossed; no Marie-Foix on either side. Clonus likewise was absent.

Concerning the motility of the limbs much doubt was felt, for although at first the patient moved the legs and arms in a manner which seemed to express conduction over intact corticospinal tracts, such movements as she made at this time were very slight and infrequent. The impression, however, prevailed that this lack of motion was due to deepening coma rather than defects in the efferent motor paths. The hypertonus observed through the earlier stages of the disease seemed somewhat to relax, the legs and arms assuming the position of full passive extension. No tremors were observed in any part of the musculature, nor were there any convulsive movements or spasmodic twitches. The deepening coma precluded the possibility of any sensory perception.

The ocular reflexes showed changes in that the conjunctival and pupillary light reactions, absent at the first examination, were now present. The pupils were less contracted and were equal in size. The eyes and head, at first so markedly turned toward the right, now manifested much less of a tendency in this direction. While the head was, for the most part, turned so that the chin was directed toward the right, the eyes were held less persistently in this direction and frequently drifted back to the primary position.

The tension in the muscles of the neck was much less than at first. The tongue now assumed a more normal position in the mouth and was moved normally in swallowing and respiration. There was no sialorrhea and such saliva as was secreted the patient swallowed with slight difficulty. Fluid given by mouth in any but small quantities caused choking and coughing. The degree of dysphagia was slight as was also the palatoptosis.

The essential changes in the neurological status at the end of nine hours consisted in:

1. A general increase of the tendon reflexes.
2. The disappearance of the abdominal reflexes.
3. The bilateral appearance of a hitherto absent Babinski.
4. The general relaxation of the muscle tone.
5. The absence of convulsive movement and spasmodic twitchings.
6. The presence of pupillary and conjunctival reflexes which were absent at the onset.
7. The less marked oculogyric and cephalogyric tendency towards the right.

The general condition remained as above noted until the early part of the twelfth hour, at which time the blood pressure rose rapidly to 270. Following this the pulse rate increased from 112 to 170 per minute. Cyanosis began to show in the face; the breathing became more rapid and shallow and a profuse general perspiration occurred. About ten minutes after the blood pressure rise, there appeared convulsive twitches, first in the face muscles and later in the arms, where the principal movement was extreme extension at the elbows with clenching of the hands. The duration of each spasm was from one to two minutes with a varying interval of three to four minutes between them. Later these extension-spasms involved also the trunk and leg musculature so that the entire body was held rigid in the extensor posture but true opisthotonos was not seen. With each spasm, the breath was held, cyanosis deepened and the pulse became more rapid and thready. These convulsions lasted over a period of about thirty minutes. Following this, the patient became rapidly weaker and more cyanotic. With the rapidly falling blood pressure, the pulse became somewhat slower, having considerably less force. Respirations grew more shallow and slower in rate, decreasing in about fifteen minutes from about 40 per minute to 4, later 2, then 1, and the last two respirations were separated by an interval of three minutes. After thoracic and diaphragmatic breathing had ceased, the patient continued to make gapping movements with the mouth as though attempting to breathe. These movements consisted of a rhythmical depression of the lower jaw attended by extension of the head. During this period, the heart maintained a rate of about 160 per minute, decreasing in force with the decreasing respiratory rate until the radial pulse was no longer to be felt, during the last five minutes. Cardiac sounds at the apex, however, could be heard distinctly for about two minutes after the last respiration.

THE AUTOPSY AND PATHOLOGICAL FINDINGS

The autopsy was performed five hours after death by Dr. John H. Larkin, to whom we are indebted for the following description:

Upon removal of the calvarium, the dura appeared very much congested and presented many areas of small, circumscribed hemorrhage. Upon opening the dura the spinal fluid escaped, having the appearance of thin, venous blood. It was under considerable pressure, which latter appeared to be evenly distributed throughout the subarachnoid space in its relation to the convexity. Upon removal of the dura, the pia-arachnoid gave the appearance of marked injection, becoming progressively more intense in proceeding down the convexity of the hemispheres toward the base. In the region of the left inferior temporal convolution and as far forward as the tip of the temporal lobe, there was a thin film of subpial hemorrhage. The convolutions of the frontal, parietal, occipital and temporal lobes appeared in normal condition, their fissures not being unduly wide, nor their surfaces edamatous or otherwise made prominent.

On removal of the brain from the skull, the entire basal region was found to be concealed by a thick layer of subpial hemorrhage as well as extensive hemorrhagic extravasations into the basal cisterns. The subpial hemorrhage had the following distribution upon the basal surface: In the orbital region of the frontal lobe on both sides as far forward as the middle orbital sulcus, surrounding the olfactory tract and the ventral portion of the gyrus rectus in the region of the lamina terminalis, dorsal and ventral to the optic chiasm; upon the entire under surface of the left temperosphenoidal region and the mesial portion of this area on the right. The basilar cistern was filled with clotted blood, thus obscuring the tuber cinereum, the mammillary bodies and the cerebral peduncles. The pontile cistern and the cisterna magna were likewise filled with clotted blood, so that the ventral surface of the pons, the occipital surface of the cerebellum, and the ventral surface of the medulla were obscured on both sides. The coagulated blood in the three cisterns formed a firm layer varying in thickness from one-fourth to one-half inch. Its greatest thickness was in the region of the basal cistern. Upon the separation of the spinal cord and the removal of the brain, the subdural space was found to contain an unusual amount of thin, bloody cerebrospinal fluid.

The blood vessels constituting the circle of Willis and the chief branches therefrom were carefully examined and with one exception presented no evidences of atheroma or other degenerative changes. The exception referred to was the *anterior communicating artery* where a small aneurysmal dilatation was found about 2 mm. from the junction of this communicating artery and the right anterior cerebral. This aneurysm was about 2 mm. in diameter and had ruptured upon its dorsal aspect. Firmly attached to the interior surface of the aneurysm was a firm, greyish mass about the size of a shelled almond and apparently the fibrinous remnant of a clot of some weeks standing. It was attached to the dorsal aspect of the aneurysm and extended from this point into the longitudinal fissure along the lamina terminalis for a distance of 1½ cm.

Upon macrotomic section of the brain, the hemorrhage was seen to have invaded the ventricular system. The anterior, posterior and descending horns of the lateral ventricles were filled by a dense and firm clot of blood which appeared to be of relatively recent coagulation. The intraventricular pressure due to the clot had caused considerable distortion as witnessed conspicuously by the compression of the corpus callosum, the head of the caudate nucleus and the optic thalamus. The hemorrhage also invaded the other ventricular

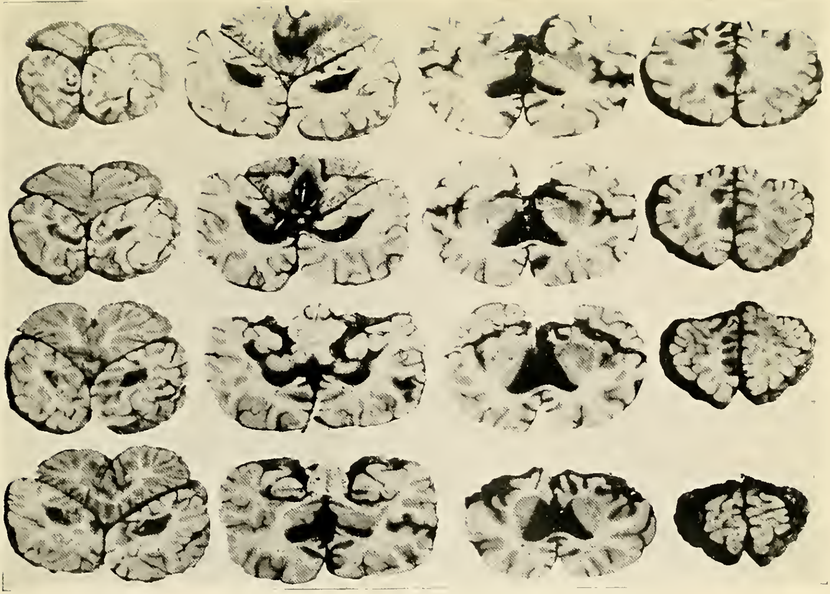

Fig. 1. General View of the Ventricular Hemorrhage in the Several Divisions of the Ventricles

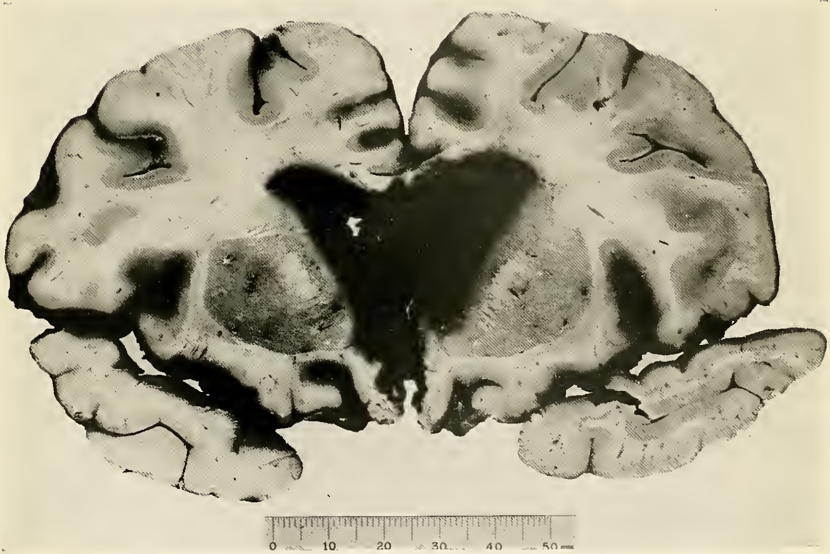

Fig. 2. Level of Junction between Body and Anterior Horn of Lateral Ventricle, Showing Compression of Caudate Nucleus and Corpus Callosum

spaces of the cerebrum causing a marked dilatation of the aqueduct of Sylvius, the third and the fourth ventricles. Macrotomic section revealed the fact that the subpial hemorrhage, which was so slight upon the convexity, was much more extensive in the fissures, particularly those of the temporal lobe and the Sylvian fissure. The brain itself showed little change other than that due to the intraventricular pressure; the centrum ovale and corpus striatum presented nothing pathological in the gross. The cerebellum, pons, midbrain and medulla were normal in configuration except for the marked distention of the fourth ventricle. The macrotomic sections taken at several levels are presented in Figures 1 to 4.

CORRELATION OF PATHOLOGY AND SYMPTOMATOLOGY

The Prodromal Period.—The explanation of the definitely prodromal period, lasting for a month or more, is, we believe, to be sought in the presence of the relatively large fibrin clot which was found attached to the anterior communicating artery. That the formation of this clot long antedated the development of the patient's massive, cerebral hemorrhage is witnessed by the nature of the clot itself. Evidently it was of some long time standing. Its hard consistency, its greyish color and its beginning organization testify to the fact that it was the result of an earlier and slighter hemorrhage. At what exact time this latter vascular accident occurred it is difficult to decide but it would seem likely that its incidence was associated with the first appearance of the psychic changes in the patient. It is conceivable that her increasing fatigability, her unusual pressure of activity, unrest and irritability, together with the irregular, fitful headache were due to the irritation of the clot upon the frontal lobes. It was undoubtedly the increasing headache due to irritation of the meninges which caused her to seek relief through an increase of her usual activities in order to reestablish her sense of well being, but struggling as she was against an irremovable cerebral irritation, her activity left unsatisfied her efforts toward relief and resulted only in fatigue and a consequent irritability.

The Onset.—With such a period of preparation it is not difficult to understand how the patient's actions immediately prior to the onset led to the profound vascular changes which ended in cerebral hemorrhage. Her rapid ascent of the stairs after suddenly arising from her bed suffices to explain the sudden and probably excessive rise of the blood pressure. The aneurysm in the anterior communicating artery, already the site of a previous small hemorrhagic leak, now gave way entirely, permitting the flooding of the basal cisterns. The increased intracranial pressure gave rise to severe pain in the head, the steady expansion of which was due to the spreading of the hemorrhage, while its wave-like character was caused by the effect of the respiration upon the intracranial pressure. The distribution of the head pain to the vertex, frontal, parietal and occipital regions was determined by dural compression producing irritation of the trigeminal nerve. The rapid extension of the pain into the neck, shoulders, hips and rectum followed directly as the result of compression upon the dorsal spinal roots and the pressure of free blood in the subarachnoid space around the spinal cord.

Evidence of Intracranial Pressure.—When the pain reached its culmination, coma supervened in consequence of the rapidly increasing intracranial pressure which in turn produced a dysdiämorrhysis. The factors leading up to this latter condition have been well summarized by Cushing (2):

> The first stage, that of circulatory compensation, corresponds to an early period or mild degree of compression, during which the encroachment on the intracranial space, by some foreign substance which displaces the normal contents of the cerebral chamber, is not sufficient to seriously compromise the circulation. By the escape of cerebrospinal fluid and by a certan amount of narrowing of the venous channels, the process is accommodated with the avoidance of any pronounced degree of venous congestion of the brain. Under these circumstances symptoms, although not absolutely latent, are in the main insignificant.
>
> If the process continues to the point of failure of this circulatory compensation a condition of venous stasis is brought about, with a lessening of the normal amount of blood flowing through the capillaries (dysdiämorrhysis). At this time symptoms of

disturbed cerebral function are inaugurated evidencing an early stage of actual compression-headache, vertigo, restlessness, roaring in the ears, a disturbed sensorium with excitement or delirium, an unnatural sleep, etc. Other symptoms also become outspoken. Among these, of highest importance is the recognition by means of the opthalmoscope of some distention and tortuosity of the veins radiating from the optic papilla; indeed, a certain degree of venous stasis may have been appreciable in the eye-grounds during the previous stage of compensation. Indications that the venous congestion is influencing bulbar circulation may be apparent as a slowing of the pulse with or without a certain rise in blood pressure.

The third stage, that of advanced compression phenomena, corresponds to a condition of capillary anæmia due to a further increase in intracranial tension. This anæmia in case of 'local compression' may have exerted its effects primarily over a circumscribed area of the cerebrum, throwing out of function that part of the brain in its vicinity with corresponding paralytic symptoms. Should, however, the pressure from such a local process be sufficiently far-reaching to involve the medulla, or should the medulla from the start have been involved in the threatened anæmia in correspondence with the rest of the intracranial contents (that is, in case of 'generalized compression'), then the period of vasomotor regulation follows with its characteristic rise in blood pressure. This reaction may last an indefinite period, until relief is afforded or until an increase in compression is more than can be compensated for, and the terminal state of compression steps in to supplant it. Associated with the rise in blood pressure may be seen marked respiratory disturbances, which are especially distinctive when the former is accompanied by alterations in level. The periodicity of symptoms so often observed during this stage, and referable not only to respiration, but shown also by rhythmic alterations in size of the pupils, by wavering increase and diminution in depth of stupor, and by many other signs indicative of alterations between irritative and paralytic symptoms, is due to this basomotor rhythm associated as it is momentarily with sufficient cerebral circulation (eudiämorrhysis) alternating with a period of inadequate or totally deficient blood supply (adiämorrhysis). Vagus symptoms are, as a rule, pronounced during this stage, the pulse being slowed to 50, 40 or even fewer beats per minute, but without, or, at all events, with much less apparent evidences of rhythm.

The terminal stage which follows upon an increase of intracranial tension over and above that which can be compensated for by stimulation of the vasomotor center, is characterized by a falling blood pressure, irregular cardiac and respiratory efforts, deep coma, which supplants the varying degrees of stupor, complete muscular relaxation, wide pupils, broken, snoring respiration, etc., soon to terminate in cessation of all cerebral function, with respiratory paralysis and with the rapid pulse of low pressure, indicating failure of the vasomotor mechanism. An irrecoverable condition of cerebral anæmia (adiämorrhysis) has been reached.

Accepting this interpretation of the vascular disturbances due to increased intracranial pressure of cerebral hemorrhage, it would appear that the patient passed almost at once into the vascular status of cerebral dysdiämorrhysis. The evidence of this is found in the disturbances in respiratory and cardiac activity. In this condition she remained for the first four hours, gradually regaining, however, some degree of respiratory and cardiac compensation, since the irregular breathing of the Cheyne-Stokes type little by little gave place to a more regular respiratory movement. The irregularities of the pulse also disappeared in the same period. This compensation we take to represent the diämorrhytic accommodation of the circulation following the shock of the original insult. This condition was maintained until the beginning of the twelfth hour when, according to our interpretation, the ventricles were flooded and the terminal stage made its appearance. During this time, after a sudden rise, the blood pressure began to fall, the cardiac and respiratory activity again became irregular, the pupils dilated and the "irrecoverable stage of cerebral anæmia had been reached."

The Blood Pressure.—It is not our intention to enter into a discussion either of the complex mechanism in the regulation of blood pressure or the disturbances leading up to hypertension in certain cerebral conditions. We believe that there was in this case a fundamental arteriosclerotic change as a result of which the patient probably ran a relatively high blood pressure prior to her last illness. Her cerebral hemorrhage undoubtedly accounted for the initial rise and, in all probability, as a result of the vagotonic stimulation, maintained the pressure at a high systolic level with a correspondingly high pulse pressure. It is to be borne in mind that from the second to the ninth hour the patient received fairly

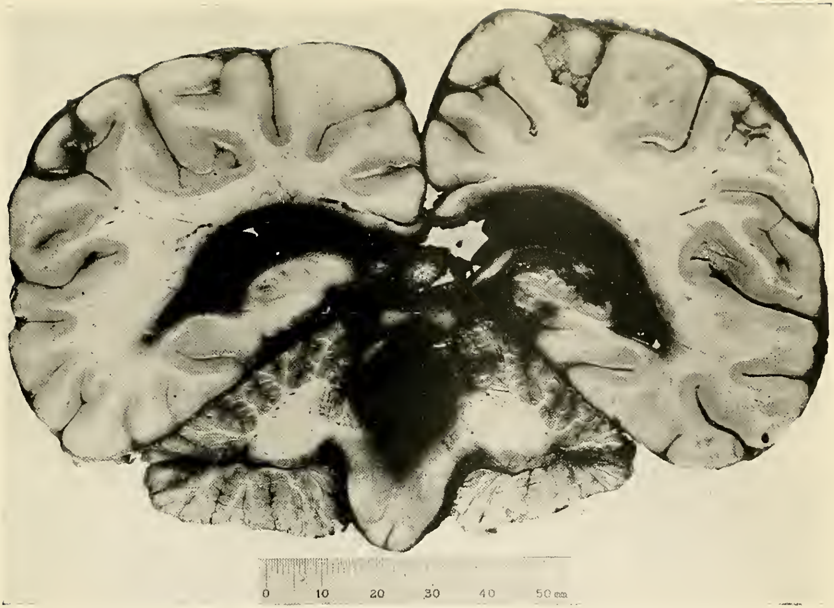

FIG. 3. LEVEL OF THE BODY OF THE LATERAL VENTRICLE, SHOWING COMPRESSION OF OPTIC THALAMUS, INTERNAL CAPSULE AND LENTICULAR NUCLEUS WITH HEMORRHAGE IN THE LATERAL AND THIRD VENTRICLES; ALSO IN THE CISTERNA BASALIS AND CISTERNA SYLVII

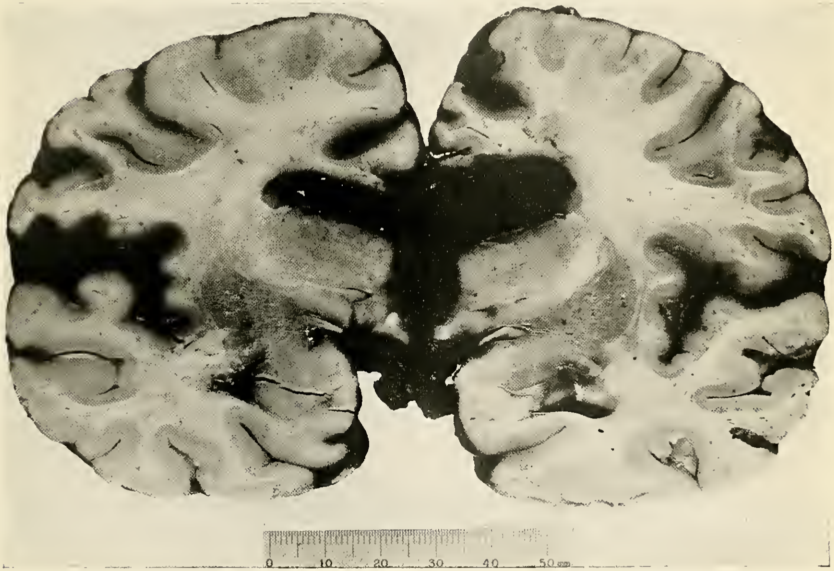

FIG. 4. LEVEL OF THE TRIGONUM VENTRICULI, SHOWING HEMORRHAGE

large doses of cardiovascular depressants (aconitine and gelseminine hydrochlorate) administered hypodermatically and at short intervals. The administration of these drugs was begun in the second hour and it was during this period that a marked drop of 20 mm. Hg. was noted, *i. e.*, from 220 to 200. A still further decrease in pressure occurred at the fourth hour and was maintained until the ninth hour. It is by no means evident that the cardiovascular depressants employed produced this decrease in blood pressure for it must be remembered that a definite compensation appears to have taken place in all the symptoms after the shock-like phase witnessed in the period of dysdiämorrhysis. In this period, which lasted for several hours (about the first four), there were many clinical signs of failing compensation in cardiac action and respiration; the chief of these were the Cheyne-Stokes type of breathing and the irregularity of the heart, both in rate and systolic pressure. In this latter connection it may be said that while the majority of cardiac cycles carried the blood through the vessels at a pressure of 200 mm. Hg. many of them were much below this level. Compensation was apparently established in the fifth hour and the patient's condition seemed more favorable. The pulse, although rapid, was regular, the respiration lost its Cheyne-Stokes character, and the blood pressure showed a further maintained reduction to 195 mm. Hg. We felt justified in the belief that the hemorrhage was at length controlled. According to our present interpretation, this marked the termination of the hemorrhage into the basal cisterns and the subarachnoid space.

Four hours later, namely, in the eighth hour, cardiovascular compensation showed signs of renewed instability. The blood pressure suddenly rose to 230 mm. and in the next succeeding hours with a number of fluctuations oscillated between 230 mm. and 200 mm. In the hours just referred to the following readings were taken (*See Chart of blood pressure record*):

9:00 o'clock	200 mm. Hg.
9:15 o'clock	230 mm. Hg.
10:15 o'clock	210 mm. Hg.
10:30 o'clock	215 mm. Hg.
11:00 o'clock	210 mm. Hg.
11:30 o'clock	200 mm. Hg.
12:00 o'clock	210 mm. Hg.
1:00 o'clock	210 mm. Hg.
1:30 o'clock	230 mm. Hg.
2:00 o'clock	220 mm. Hg.
2:30 o'clock	270 mm. Hg.

This irregularity and general tendency toward elevation is in marked contrast to the conditions in the hours immediately preceding. In our opinion this change indicated the development of the second phase in the cerebral hemorrhage, namely, the invasion of the ventricles. Breaking through the lamina terminalis and replacing the spinal fluid, the blood stimulated the nerve elements. Slowly the ventricles became filled, then distended under greatly increased pressure. This new element acted first as an irritant to the cardiovascular centers which finally, as a result of the unremitting pressure, became compressed and ceased to act.

The Respiratory Disturbances.—During the first four hours the breathing was Cheyne-Stokes in type. Thereafter it lost this character and, like the cardiac activity, manifested evidence of compensation until the thirteenth hour. This observation is significant in view of the statements made by several observers, including Cushing, Eyster and Pollock, to the effect that Cheyne-Stokes respiration is frequently associated with increased intracranial pressure. In the main these statements are correct, but they fail to call attention to the fact that even suddenly increased intracranial pressure of relatively short duration may permit of compensation on the part of the compromised brain tissue. There can be no question as to the increased intracranial pressure in this case, nor can there be doubt that it produced a constant compression. Yet in spite of this, the brain, withstanding the first assault, subsequently adjusted its respiratory mechanism to such a degree that the Cheyne-Stokes movement disappeared. Another interesting feature concerning Cheyne-Stokes respiration first noted by Cushing and later confirmed by Eyster is the fact that in this type of breathing due to increased intracranial tension, the blood pressure is low during the apneic phases and high during hyperpneic phases. In this particular the Cheyne-Stokes respiration due to intracranial conditions differs from that caused by such diseases as general arteriosclerosis, mitral insufficiency myocarditis, infarct of the lungs and nephritis. In these latter disturbances the blood pressure is high in the apneic phase. Such fluctuations of blood pressure as were noted in the first four hours of this case showed a tendency for the pressure to decline five to ten points during apnea. This confirms the observations made by the previous observers quoted with reference to the Cheyne-Stokes breathing due to increased intracranial pressure.

One other respiratory phenomenon not pertinent alone to cerebral hemorrhage, but frequently noted shortly before exitus in many diseases is worthy of passing mention. It is referred to in our case as follows: "The respirations grew more shallow and slower in rate. After thoracic and diaphragmatic breathing had ceased, the patient continued to make gapping movements with the mouth as though attempting to breath." May there not be a phylogenetic reason for these final mouth movements? Is not, perhaps, the ancient history of the respiratory mechanism making itself manifest in these last, pertinacious movements of the jaw which hark back to the important rôle of the mouth in the gill breathing of aquatic animals? Such an hypothesis is at least suggestive, for it seems to indicate, as might be expected, that the most ancient part of the central respiratory mechanism in man, namely, that controlling the oral movements, is last to succumb on the approach of death.

The Convulsive Movements.—The spasms of the muscles which almost immediately followed the onset of coma definitely affected the cephalogyric and oculogyric systems since the head and eyes were turned to the right. According to Binswanger's interpretation (1) of spasmodic and convulsive activity, tonic spasm is due to the inhibited action of the midbrain, pons and medulla. If such a theory is at all tenable then it must have been the case in our patient that the excessive irritation of the brain stem caused the spasmodic contractions of the neck and eye muscles. The cephalogyric and oculogyric fibers would, in all probability, be most exposed to such pressure in the region of the cerebral peduncles where the pes profundus and superficialis convey those aberrant pyramidal bundles which establish the corticonuclear connection for the sixth and eleventh cranial nerves.

The arm, trunk and leg spasms, also essentially tonic, might in the light of this interpretation be explained by the irritation of the brain stem by free blood in the basal cisterns. The tonic condition of the entire body, including the spasms of the extremities and neck and eye muscles, subsided during the fourth hour due to the effects of overstimulation of the brain stem.

In the terminal stage a new series of convulsive movements made its appearance which differed in a marked way from the spasms of the initial phase. These spasms were wholly tonic in character. They produced a dominant extension in the trunk and neck in orthotonos while the extremities, with the exception of the fingers which were held in hypertonic flexion, were held extended and adducted. Each spasm lasted one to two minutes, the patient being quite relaxed for an interval of three to five minutes between attacks. After a period of thirty minutes, these convulsive movements ceased and the patient grew rapidly weaker. These spasms we take to indicate the final extension of the hemorrhage into the lateral and third ventricles while their tonic nature suggests the possibility of an involvement by pressure of the basal ganglia adjacent to these cavities, that is, the optic thalamus and corpus striatum.

The Visceral Disturbances.—The vomiting, defecation and urination which occurred with the onset of coma were undoubtedly due to the initial stimulation of the bulbar autonomic which produced the emesis, and of the pelvic autonomic giving rise to the forceful defecation and urination. The direct cause of these disturbances was pressure upon the vagal and spinal roots.

The Reflexes.—The observations of the first neurological examination showed that the deep and superficial reflexes were normal. This might be explained by the fact that the corticospinal fibers of the pyramidal system had not as yet come under the influence of pressure sufficiently to produce changes in their function. The general hypertonus, on the other hand, which at this period was not extreme, gave evidence of the beginning involvement of the pyramidal tract. The later appearance of a bilateral Babinski and the absence of all abdominal reflexes indicate the final involvement of the brain-stem which eventuated in compression of the pyramidal tracts by the hemorrhage.

The Choked Disc.—The choked disc of one diopter on either side was the direct result of the increased intracranial pressure caused by the hemorrhage.

The Myotonic Changes.—The general relaxation in the muscle tone and the disappearance of the tonic spasms were interpreted as the result of decreasing activity of the brain-stem consequent upon overstimulation. The contracted pupils observed during the first neurological examination later became considerably dilated and the formerly absent pupillary reflex made its reappearance. In this light the latter reflex phenomenon may be regarded in the same category as the initial hypertonus in the somatic muscles. In the case of the latter, the hypertonus was manifestly an irritation phenomenon and so likewise was the immediate contraction following the onset of the hemorrhage. Later as the result of overstimulation, this sphincteric hypertonus relaxed and the pupil became active to light stimulation.

The Temperature.—The slight sustained elevation of temperature was similar to that frequently seen in cases of cerebral hemorrhage. This phenomenon is usually attributed to the toxic effects of the liberated blood, a supposition which would seem particularly pertinent in this instance. However, the fact must not be overlooked that the area of the tuber cinerum has been said to contain certain thermotactic centers. Such being the case the presence of blood, either because of the increased pressure which it may exert upon this region or because of its irritant effects, might be sufficient to produce a rise of temperature.

Eventually when the hemorrhage had extended into the fourth ventricle, the respiratory center became progressively compromised until death ensued due to complete failure of respiration and the destruction of the other vital centers.

CONCLUSIONS

I. From the correlation of the clinical observations with pathological findings, the cerebral hemorrhage in this case had two fairly distinct phases in its development, namely, (1) hemorrhage into the basal cisterns and subarachnoid space, and (2) hemorrhage into the ventricular system.

II. Although the evidence of a single case may not suffice for the establishment of all of the symptoms in a given syndrome, the conditions in this instance were under such close and continuous observation, while at the same time they permitted unusually satisfactory opportunity for the correlation of clinical and pathological data, that we believe it is now possible, in the light of these facts, to enumerate the symptoms which result from hemorrhage into the basal cisterns, later complicated by extension to the ventricles.

III. The symptoms of hemorrhage into the basal cisterns are:

1. Sudden, intense pain in the head, having a wave-like character, rapidly increasing in severity and accompanied by severe pain extending from the neck to the shoulders, hips and rectum. This pain lasts for several moments when the patient suddenly becomes comatose.
2. Propulsive vomiting followed by forceful, spasmodic defecation and urination. This occurs shortly after the establishment of coma and may recur at intervals for several hours.
3. Tonic, sustained cephalogyric and oculogyric spasms.
4. Tonic contraction in the arms and legs accompanied by muscular spasms producing rhythmical abduction and adduction of the upper and lower extremities. These convulsive movements are synchronous with respiration and persist for several hours.
5. Rapid, irregular cardiac activity with high, fluctuating blood pressure. Cheyne-Stokes respiration. In the course of several hours these cardiac and respiratory symptoms cease.
6. No initial change in the reflexes but later a loss of the superficial reflexes, an increase

of the deep reflexes and the appearance of a bilateral Babinski.

7. Moderate degree of bilateral choked disc —*i. e.*, one to two diopters.
8. Slight rise of temperature—*i. e.*, 100°–101°.

IV. The symptoms indicating extension of the hemorrhage to the ventricles are:

1. Repeated tonic spasm of the entire somatic musculature, holding the trunk and neck in orthotonos, the extremities extended with exception of the fingers, which are flexed.
2. These spasms last for one to two minutes and are followed by an interval of complete relaxation.
3. Marked elevation of the blood pressure followed by a rapid decline.
4. Irregularity of cardiac and respiratory action.

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DISCUSSION

DR. WILLIAM M. LESZYNSKY. Taking into consideration the high intellectuality of the patient herself, and the pathological correlation of the symptoms, this case presented unusual facilities for thorough clinical observation.

The statements made by Dr. Tilney recalled several things to my mind that occur in connection with attacks of cerebral apoplexy. The general practitioner has many opportunities to see patients during the period of an acute attack, but he has rarely been present from the very beginning. Some years ago I watched the development of the classical symptoms of hemorrhage into the pons. The patient was a very intelligent woman fifty years of age, who had been under the care of her physician for chronic nephritis. She arose one morning with headache and numbness on one side of her body. I found her with speech unimpaired and no disturbance of consciousness, but complaining of headache, vertigo and diplopia. There was facial and abducens paralysis on the same side. Within one hour she gradually became comatose, with complete hemiplegia upon the opposite side, pinhole pupils, temperature 106 degrees F., and a very high blood pressure. When a vein in the arm was incised the blood squirted at least six feet across the room. The coma increased until death supervened in about twenty-four hours. There was no autopsy.

I remember another case of a man weighing over 200 pounds, who had an attack of cerebral hemorrhage during which the systolic blood pressure was over 260. He was comatose and about sixteen ounces of blood were withdrawn, whereupon the pressure was reduced to 180 and he seemed to improve for a time, but in four or five hours the pressure went up again to 260. Another phlebotomy was done but it did not seem to have more than a temporary effect. I have witnessed the same procedure and result in a number of instances, and have come to the conclusion that as an emergency measure it accomplishes a reduction of blood pressure more satisfactorily than medicinal therapy.

MULTIPLE SCLEROSIS ASSOCIATED WITH INTERNAL HYDROCEPHALUS

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This case warrants presentation, especially for the reason that, in addition to the usual manifestations of disseminated sclerosis, clinical evidence of intracranial lesion of vague character obtruded itself into the picture of what appeared to be a well-defined case. Occurring several years after the apparent onset of the symptoms of multiple sclerosis, the cerebral symptoms of uncertain etiology at the time did not lend themselves to proper interpretation. A variety of convulsive seizures led to the supposition that either fresh hemorrhages had occurred in the upper motor neurone, probably in the cortex, or that the pathology of the clinical manifestations was determined by sclerotic patches in the cerebrum. Autopsy findings, a brief account of which will be presented, clearly indicate a definite lesion of another nature giving rise to the diverse symptoms.

In this brief account we shall not attempt to discuss the relationships between clinical manifestations and pathological findings in multiple sclerosis. Cases such as the one presented suggest the probability that the bizarre and varying symptoms covered by the term "multiple sclerosis" may at times have as their cause, not only the usual disseminated characteristic patches, but either antecedent or concommitant changes of a different nature in the central nervous system. Indeed, we are not infrequently impressed by the recession of most severe manifestations in the course of our observations of cases of multiple sclerosis.

It is difficult to reconcile the sometimes sudden and most striking returns to almost normal function with the typical pathology of multiple sclerosis, which would seem to preclude anything like complete return to physiological function. As a matter of fact, there are cases at the Montefiore Hospital, which have been under observation for some time, that present symptoms justifying without question the diagnosis of multiple sclerosis, and which have in addition evidence of involvement of the nervous system of a character other than that found in multiple sclerosis.

In the light of the observations here recorded, we expect to present at the Conference in the near future, a similar case of disseminated sclerosis, occurring, however, in a far younger patient, in which many of the symptoms are doubtless due to internal hydrocephalus.

Patient: The subject of this presentation, E. D., was a male, aged 48 at death, born of French parentage, in the United States.

HISTORY. Of importance in the history of the patient are the facts that he was moderately indulgent in alcohol and tobacco, and that so far as it is possible to determine by the history obtained and repeated findings of spinal fluid and blood, he did not suffer from syphilis. The only acute infection was a pneumonia, suffered three years, however, subsequent to the onset of the nerve symptoms and from which the patient made a complete recovery.

The record shows that the patient appeared in perfectly good health until his thirty-second year. At this time (1902) he fell from a car, striking his back. The history does not show that this fall was the result of defect of the functions of the nervous system, such as defective equilibrium or sudden loss of

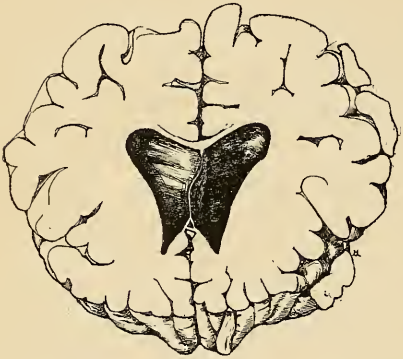

FIG. 1. FRONTAL SECTION OF CEREBRAL HEMISPHERES

Shows marked dilatation of anterior horns of lateral ventricles

power; it appears to have been in the nature of the usual accident. No immediate symptoms followed, the patient being able to continue his work. He did not suffer with pain or restriction in locomotion or equilibrium. On the following day, however, some vague pains in the back and weakness of the lower extremities developed, and we have a history of his confinement to his home for one month. The details of this period of illness were not supplied, but he gradually recovered entirely. Some five years later there gradually again developed disturbance in the use of the lower extremities, apparently as paresis.

A second fall some few months later was due to the condition of the patient but made no change in the physical symptoms. About six months later, *i. e.*, in 1908, the patient was admitted to the Montefiore Hospital with the following complaints: weakness and stiffness of the lower extremities, occasional vertigo, some difficulty in controlling sphincters. (It is to be regretted that the routine examination such as is now regularly in use in the Neurological Department of Columbia, was not at that time instituted. Routine detailed observation was not followed in the examination; hence much important data are not at our disposal.) There were no abnormal attitudes. Gait was extremely spastic with marked hypertonia of the lower extremities. At this time the upper extremities showed no abnormalities other than that there was marked exaggeration of all the deep reflexes. The lower extremities showed all the features of upper motor neurone type of paralysis, the spasticity more marked in the right than in the left leg, double Babinski with all its confirmatory signs—all these much more marked in the right than the left leg. The abdominals were feeble, those on the right side being hardly elicitable. The cranial nerves were all carefully examined, and other than marked nystagmus on lateral fixation were recorded as normal. Speech is described as suggestive of the scanning type. Peculiarly enough, the optic discs examined by a competent attending oculist are recorded as suggestive of syphilitic vascular change. There was absolutely no other suggestion of this kind throughout the course of the disease, and a somewhat later examination of the eyes gave as a definite finding only bitemporal pallor.

Records of the hospital show practically no change in the patient's condition for the next three or four years.

In 1913, anisocoria was recorded, with somewhat sluggish reaction to light and accommodation; furthermore, there was temporary weakness in the right upper extremity, loss of the appreciation of deep sensibility and atrophy of the interossei. Further, tremor of the

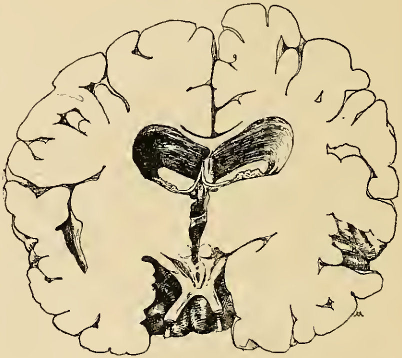

FIG. 2. MARKED DILATATION OF BODY OF LATERAL VENTRICLES; ALSO THICKENING OF EPENDYMAL LINING

head in the upright position, a tremor of the intention type in both upper extremities, also intense emotionalism with a tendency toward uncontrollable outbursts of laughter were noted.

The next important observation recorded is in May, 1914—some eight months later. At about midnight, during sleep, a generalized convulsion was recorded with complete unconsciousness and dilated and sluggish pupils. In December, 1914, a similar generalized convulsion with complete unconsciousness occurred. These convulsions were followed by a short stage of stupor, but further details are not available. In May, 1915, a convulsive seizure of a different character was observed. The patient was found unconscious, the head and eyes turned to the right, the right forearm flexed in tonic contraction, clonic seizures of both sternocleidomastoids with alternate rotations of the head. There followed a transitory paresis of the right side of the face. Variability from time to time in the size and reaction of the pupils at this period of the history is recorded.

On the day of the convulsive seizure last described, the patient was stuporous and on recovery somewhat confused, with irregular pupils—the left larger than the right—both markedly dilated. At this time was also noted spasticity of both upper and lower extremities with marked exaggeration of all deep reflexes and absence of the abdominals. The cloudiness of mentality which succeeded this convulsion cleared up only after some three weeks. Examination about a month later showed marked bitemporal pallor.

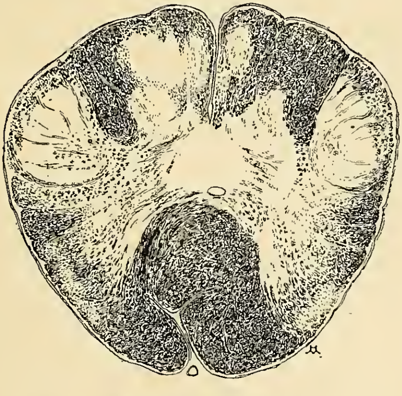

FIG. 3. SECTION OF MEDULLA AT LEVEL OF PYRAMIDAL DECUSSATION

Shows sclerotic patch involving the tractus gracilis and tractus cuneatus, partially involving the nucleus gracilis on right side

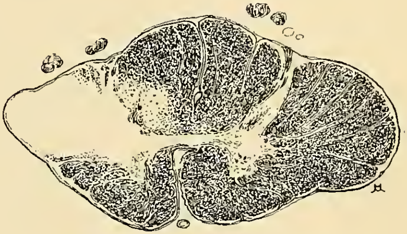

FIG. 4. SECTION OF SPINAL CORD AT LEVEL OF SIXTH CERVICAL SEGMENT

Shows sclerotic changes involving all tracts included in lateral column and major portion of anterior horn on right side

We have a history of seizures for the next few months, most variable in character, but with markedly greater involvement on the right side. The spasticity likewise was subject to variability. The usual signs of pyramidal tract involvement were markedly increased in the right leg during the convulsive seizure; the right big toe, during these seizures, would be held in extreme extension, a confirmatory Chaddock very easily elicited, and slight stimulus induced ankle clonus. In fact, it is of interest to note that the convulsive seizures were comparatively mild on the left side of the body and most active on the right. During one set of seizures, the attacks were repeated at irregular intervals for twenty-four hours. Observations made at this time showed that just previous to an attack the blood pressure would suddenly rise. Taken during one seizure it was found to be, systolic 220, diastolic 150. The normal blood pressure of this patient varied between 108 and 130 systolic, and 80 and 90 diastolic.

On the day following one seizure the patient was found to have motor aphasia with what is described as a hemiplegic position of the right arm and leg. Lumbar puncture at this time showed no increase in pressure and, as heretofore, negative serology.

During 1916, generalized convulsive seizures followed by stuporous states, with only transitory spasticity in the right upper extremity, were recorded. In December, 1917, marked diminution of joint, muscle, tendon sense in the lower extremities, was recorded.

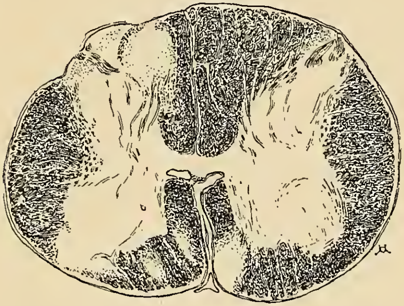

Fig. 5. Section of Spinal Cord at Level of Fourth Lumbar Segment

Shows patches in right dorsal and both ventral columns, more marked on right

In January, 1918, after varying periods of delirium and stupor, the patient died.

Family History.—Other than a uniform history of vascular disease both on the paternal and maternal sides, the family history contributes no etiological factor.

AUTOPSY FINDINGS

Nervous System. *Brain.*—The calvarium was removed with difficulty because of dural adhesions. These extend over an area corresponding to a zone of 6 cm. in width on either side of the dorsolongitudinal fissure. On separating these adhesions the brain is freed and is found to be of normal size, hemispheres being equal. The arachnoid covering the dorsolateral surfaces of the hemispheres is somewhat opaque and markedly thickened along the dorsolongitudinal fissure. The gyri are somewhat less prominent than normal, the sulci being somewhat shallower than normal. The meninges at the base are normal. The cranial nerves at their origin are apparently normal. On making a frontal section the ventricles are disclosed and are found markedly enlarged. The ependyma lining these ventricles is definitely thickened and the septum lucidum is extremely thin and fenestrated. The cortex is somewhat reduced in volume, basal ganglia showing no definite pathological changes which can be detected by the naked eye.

Spinal Cord.—The spinal cord is markedly diminished in circumference throughout. The cut section shows degeneration of the dorsal tracts and of the crossed pyramidal tract on the right side at various levels.

MICROSCOPICAL SECTIONS

Figure 1 presents a frontal section of the cerebral hemispheres, showing marked dilatation of the anterior horns of the lateral ventricles.

In addition to the marked dilatation of the body of the lateral ventricles, there is presented also in Figure 2, the very manifest thickening of the ependymal lining.

A section of the medulla at the level of the pyramidal decussation is presented in Figure 3. It shows a sclerotic patch involving the tractus gracilis and tractus cuneatus, partially involving the nucleus gracilis on the right side.

Figure 4 presents a section of the spinal cord at the level of the sixth cervical. Sclerotic changes involving all of the tracts included in the lateral column and the major portion of the anterior horn on the right side are shown.

A section of the spinal cord at the level of the fourth lumbar segment is illustrated in Figure 5. Patches are shown in the right dorsal and in both ventral columns, more marked on the right.

SUMMARY AND DIAGNOSIS

There was no evidence of sclerotic patches in the cortex. The etiology of the ventricular dilatation in this case is speculative. It appears to us quite likely that it was the result of an ependymitis. Its association with multiple sclerosis in this case, might have been purely coincidental, but it is much more likely that the pathology of the two conditions is identical. The hydrocephalus may have been of a purely obstructive type though there was no evidence of obstruction in the Sylvian aqueduct. It is possible that lesions of multiple sclerotic character in the neighborhood of the ventricles may have determined a relatively temporary occlusion of the Sylvian aqueduct, mechanically causing internal hydrocephalus. The convulsive seizures, the pupillary changes and stuporous states were all doubtless due to variation in the intracranial pressure determined by the amount of fluid in the dilated ventricles.

It is an interesting observation that the convulsive seizures and spasticity were more marked in the right half of the body and that the pyramidal tract irritability during the seizures was more marked on the right side, in spite of the fact that the pyramidal tract lesion was largely confined to the right side of the cord.

One or both of two interpretations seem probable: We know that microscopically the sclerotic areas in frank lesions of multiple sclerosis consist of proliferated glia, in which may be found remnants of degenerated nerve cells and fibres. However, the axones may remain, the damage to them being of limited extent, only the medullary sheaths being lost early in the process. Indeed, this loss of medullary sheaths may be the only result of the early process. In the case before us, we know that the sheaths of the axis cylinders in the right pyramidal tract, at the level of the cervical enlargement, have been destroyed, and it is probable that the axis cylinders themselves are only in part involved. Thus stimuli from the cortex might still pass through, giving more diffuse and intense reaction. If, on the other hand, the inhibitory influence of the cortex through the pyramidal tracts in the lateral column is removed by complete destruction in the cervical region, then the impulses passing through the direct pyramidal tracts in the ventral column might produce a more intense reaction.

The observations on joint, muscle, tendon sense, and atrophy of the interossei in the right upper extremity are readily explained by the involvement of the dorsal column (right side), and findings in the anterior horn cells. A more detailed histological study of the brain and cord is being made, in search of further involvement by the sclerotic process.

INFANTILE PSEUDOBULBAR PALSY WITH CHOREO-ATHETOSIS*

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The case is presented as one exhibiting a rather unusual combination of the symptoms frequently seen in the chaotic group of infantile paralyses.

Patient: L. F.; No. 17,045; male; born April 20, 1909, and is therefore now eight years and eight months old. He is a Hebrew and was born in the United States.

History. *Present Illness, beginning with Complaint.*—The chief complaint is speech defect, dating probably from infancy, dribbling of saliva and peculiar involuntary movements. Those who have been with the boy are positive that his disorder is getting worse. He complains of frontal headache from time to time, which may possibly be due to hyperopia. Further details are given below.

Previous History.—Nothing is known of his birth. He is an orphan and was adopted at eighteen months. He could walk then and was just able to babble. He has never learned to speak well but understands well.

The following additional details were collected by Miss Jones, Department of Child

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* Presented at the Clinical Conference, Dec. 17, 1917

Placing, State Charities Aid Association, in November, 1917:

Aug. 30, 1909: The patient when four months old was placed at Bellevue Hospital. He had enteritis and was cured. The mother was notified but did not call for him.

Oct. 9, 1909: The patient was committed to the New York Nursery and Child's Hospital. Physical condition good; mental condition poor.[1] No deformity or abnormal condition.

June 6, 1912 (patient three years and two months old): The New York Nursery and Child's Hospital had him examined, including a blood test. He was pronounced in perfect physical condition. Miss Holmes considered him dull and not mentally up to the average child of his age, but not feeble-minded.

Nov. 13, 1914 (patient five years and seven months old): The impediment in the patient's speech was noticed. He was considered dull. He was in a free home and had good care. Was always in good health. The defect in speech grew worse[2] year after year. Drooled constantly. The boy was affectionate, tractable and made serious efforts to improve.

March 28 to April 6, 1916 (nearly seven years old): The patient was in the care of Dr. Laurans at the Post Graduate Hospital, under observation. Was etherized and the larynx examined. The doctor reported that nothing could be done for him.

April, May, June, 1917 (eight years old): The teacher reported that Leon is an extremely nervous child[3] and has a speech defect which prevents his progress in writing and reading. He seems very anxious to get along; he tries very hard, and were it not for the above mentioned misfortune, would probably rank very well with the class. He shows excellent care and seems happy and contented.

The foster mother says he has always drooled since seventeen months old, but not before. The peculiar movements have only been noted, according to her, in the last year.

(End of report by Miss Jones)

Diseases, Injuries, etc.: No additional data, except there is no history of enuresis. No history of abnormal laughing or crying.

General Physiological Status: Appetite good; bowels regular.

Family History.—No details are available. The mother was reported by the Department of Public Charities, August 27, 1909 to be ill—heart trouble and anemia—and has not been heard from since August 30, 1909. The father is dead.

While the above history shows discrepancies it is evident that the disorder dates back to infancy.

VOLUNTARY MOTOR SYSTEM. *Abnormal Attitudes and Deformities.*—Nothing noted.

Gait.—Normal, including Fournier's tests, except for an occasional involuntary movement of the character noted below, and perhaps occasional external rotation and adduction of the right foot when walking fast.

Coördination.—Normal with the above exception as to involuntary movements.

Skilled Acts.—Same as above. A slight apraxia is a possibility (*see below*). Speech is bulbar (*see* Cranial Nerves).

Reflexes: (*a*) *Deep.*—These are normal except the Achilles which cannot be elicited readily on account of hypertonus.

(*b*) *Superficial.*—These are normal; possibly rather less than usual. The abdominal muscles get tense quickly when these reflexes are tested. Occasional Babinski-like movements were noted, especially on the left.

Abnormal Involuntary Movements.—These are present. They are choreo-athetotic in character, absent in repose, and increased by muscular efforts of various kinds. They do not show the definite linkage of regular associated movements; they are most marked in the fingers, hands, face and toes, and are present on both sides.

Muscle Strength.—This is normal. Head movements and those of all of the limbs were tested, except the individual fingers and toes.

Muscle Status.—Volume and contour were normal.

Tonus: There is hypertonus, apparently most marked in legs and feet. This is seen in making passive movements and is apparently of the character of a spasmus mobilis, for complete relaxation can be secured, but the patient's coöperation in securing this cannot apparently be carried out promptly by him, though intelligently attempted.

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[1] This observation may not have much value at this early age, especially in view of subsequent developments.

[2] That the defect grew worse is questionable; it may simply have grown more noticeable.

[3] This is probably a lay characterization of the abnormal involuntary movements.

Electrical reactions: Not taken.

Muscle irritability: Not tested.

Abnormal Associated Movements.— None were noted, except those of the more general character mentioned above.

NERVE STATUS.—Not investigated.

GENERAL SENSORY (not exhaustively tested). *Touch.*—Normal, including localization.

Pain.—Normal (hypersensitive?)

Temperature.—Normal.

Vibratory.—Normal on wrists, shins and ankles.

Pressure.—Normal.

Muscle-tendon.—Normal.

Stereognosis.—Normal in the hands.

Barognosis.—There seemed at first to be a difference between the two hands in the appreciation of weight, the same weight feeling heavier in the right. A repetition of the tests made this doubtful.

CRANIAL NERVES AND HEAD. *Olfactory and Nose.*—Not tested.

Optic and Oculomotor Apparatus.—Normal except for hyperopia and astigmatism in both eyes, but more especially in left. This may account for the headaches.

Trigeminus and Mouth.—Lateral jaw movements limited, especially to the right. Up and down movements normal as to extent and strength (*see also* Vagus). Touch normal on tongue.

Facial and Face.—Voluntary control defective as tested in showing the teeth (he cannot do this well), puffing the cheeks, whistling, elevating the forehead. Unilateral control of the face is poor. The face contracts more on the left side. He smiles normally and about equally. The lower lip is protruded and there is drooling of saliva. Attempts to execute the above movements elicit involuntary movements, grimaces, more marked on the left. Taste on the anterior part of the tongue seems to be normal.

Acoustic and Ear.—Hearing is not very acute.

Glossopharyngeus, Vagus, Pharynx and Larynx.—The vocal cords are normal, phonation is normal, or nearly so. The pharyngeal reflex is present (sluggish?). Swallowing is said to be somewhat difficult; the boy is said to gulp. Whether there is a true dysphagia is rather doubtful (*see* Hypoglossus).

Hypoglossus and Tongue.—The patient can protrude his tongue, but cannot make upward or lateral movements. Passive movement to the left is possibly easier than to the right. In eating, particles of food remain in the mouth and this inadequacy of the tongue may cause the apparent dysphagia. The continued presence of the food apparently is not especially noticed by the patient, though touch is normal. The principal speech defect is the pronunciation of linguals. There is no atrophy of the tongue.

MENTAL STATUS.—The mental status appears to be normal, allowing for the patient's physical handicaps.

CRANIAL MORPHOLOGY.—Normal. The head is rather small.

SYSTEMIC. *Tegumentary.*—Normal.

Glandular.—Normal.

Cardiovascular.—Normal.

Respiratory, Gastro-intestinal, Genito-urinary and Skeletal.—These were not especially investigated but were apparently normal.

LABORATORY TESTS. *Wassermann.*—This was probably negative, but a somewhat doubtful finding made it advisable to try a provocative which proved negative.

Lumbar Puncture, Blood Count, Urine, and X-ray. Not taken.

DISCUSSION

In discussing the above findings, the first question that arises is whether we have a "true" (*i. e.*, lower motor neurone) bulbar palsy. This can be definitely excluded by the absence of lingual atrophy and by the presence of the abnormal involuntary movements, which point to a higher lesion.

The next question that arises is whether we have any paralysis at all or simply an apraxia and choreo-athetosis. It seems to me that the inability to perform such a simple and fundamental movement as a lateral tongue movement can only be due to a true paralysis. Furthermore, the hypertonus and choreo-athetosis are symptoms allied with paralysis of the upper motor neurone type.

When we attempt to decide whether we are dealing simply with a light case of double athetosis or whether it should be designated as above, it appears to me we enter a region where the distinctions are more or less arbitrary. If a double athetosis (and the distinction between this "athetose double" and the ordinary athetosis is not entirely clear to me) be regarded as excluding a true paralysis, the former, on the above grounds, should be excluded. We, however, pass into a clinical field characterized by a mixture, in varying proportions, of pyramidal symptoms (diplegias, pseudobulbar palsy, etc., with Babinski, overactive deep reflexes and spasticity), of spasmus mobilis, athetosis (which may also have a choreiform character) and apraxia, together with mental defects. Apropos of the bulbar paralysis, H. Vogt, in Lewandowsky's "Handbook," under cerebral infantile palsy, makes a distinction between a "paralytic" (flaccid?) and a "spastic" form. Under the former, he says:

"The paralytic form of infantile pseudobulbar paralysis shows in its classical form the following symptoms: paralysis of the muscles of the lower and especially the middle facial, seldom the upper. The most frequent and profoundly affected muscle is the orbicularis oris, which may alone be affected. Paralysis of the tongue and jaw muscles (masseters and pterygoids); the principal disturbances here are difficulty of chewing and deglutition, with also a difficulty of facial movements, especially of the mouth—sucking, pouting the lips, whistling, puffing the cheeks are not possible. Naturally the extent of these depends upon the degree of paralysis. Complete glossoplegia is occasionally observed. It is characteristic that showing the teeth (grinning) is almost always possible, the upper lip being in general more mobile than the lower. Lateral movements of the tongue and jaw are usually more affected than the sagittal.

"Speech is usually severely affected—usually monotonous, nasal and drawling. To the purely bulbar disturbances, disturbances of sound production due to the facial are often added. The pure bulbar phenomena (speaking, sucking, swallowing, chewing) may alone represent the disease and they constitute, well developed, its essential nature. There is also a series of other phenomena almost always present—the open mouth with great outflow of saliva, often with the tongue hanging out. In those muscle regions which show difficulties of movement there are also often athetoid movements, especially in the face, often also in the tongue. These movements are either present during repose or they first appear in attempts to speak, to show the tongue, etc. Swallowing is only to a certain degree affected; the food lodges under the tongue or in the back of the cheeks where it cannot be dislodged with the tongue. When pushed back with the finger behind the uvula it is swallowed well.

"Voluntary movements of the affected muscles are not possible but the mimetic and reflex movements are undisturbed; laughing, play of expression function well. In chewing and swallowing the disturbance is less than one would expect from the paralysis, the speech being most affected. Subluxation of the lower jaw is often present. There may be trismus and exaggeration of the masseter reflex—the only spastic symptom. Paralysis of the uvula and lack of reflex are frequent.

"There is also a series of accessory symptoms which may be present in varying degree. Compulsive laughing and crying are occasionally but not often present. Disturbances in inspiration and expiration and sometimes sighing may be present, the latter all allied with athetosis. Tachycardia is rare. There may rarely be eye muscle disturbances. No muscular atrophy.

"Extremities vary. . . . In a not inconsiderable number of cases they may be unaffected.

"In many cases there may be convulsions ushering in or accompanying the disease, especially where there is an encephalitis; also diminution of intelligence. Disturbances of growth may be present but are not characteristic.

"There may be aborted forms. The disease may appear in early youth or early in childhood. The course of the disease is very variable. In some it remains throughout life as it appears in the first weeks of life. In others, and this is especially true of those cases which arise later, the disease assumes a chronic character and gradually gets worse. Changes in the fully developed disease do not usually occur. The character of the symptoms does not change in the course of the disease. As a cause of death it scarcely comes into consideration. There are cases, though, in which the disease, after having become stationary, progresses again after an illness with fever."

When the question as to the localization of the lesion is taken up we are immediately, it seems to me, in difficulty, owing to our defective knowledge as to the anatomical lesions which produce certain of the symptoms. If the foregoing conclusions be correct, we must assume that the corticobulbar pathways for voluntary (acquired) movements are affected either in cortex, capsule, or peduncle.

Regarding the involuntary movements, it always seemed to me that there are in general alternative possibilities, neither one of which can be definitely chosen. Their frequency in infantile palsies seems to indicate that they belong to a mechanism performing the incoördinated, tentative movements of early life antecedent to established pyramidal control. In this case they would arise when there is a lesion of the pyramidal system in early life. But why not always? Those who argue that they are performed by subcortical centers, on account of lesions in the latter being found in such cases, come near to falling into a logical error inasmuch as in such cases the movements should be *abolished*—an error perhaps only partly avoided by supposing that the normally executed movement is a synthesis of the two (and other) components.

The intimate association of atheotosis with spasticity and spastic paralysis and the frequent occurrence of intelligence defects in many cases, speaks rather for a cortical disturbance accounting for it all. In the present case there appears to me to be no intelligence defect; whether there is some apraxia is perhaps doubtful. It seems to me that the involuntary movements, paralysis and spasticity, would account for any phenomena that resemble it. By apraxia here is rather meant a lack of eupraxia, the latter never having been attained. Such a condition might readily arise in cortical lesions not involving directly the pyramidal system, and might practically almost amount to a paralysis.

If we attempt to place the lesion in any subcortical region, we immediately find considerable difficulty on account of the limited paralysis, but hardly more, perhaps, than any other of the pseudobulbar palsies, which are usually supposed to be due to multiple lesions. A lesion in the cortex would not encounter these difficulties.

AMYOTROPHIC LATERAL SCLEROSIS*

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Patient: S. P.; Case No. 20,006; age 54; male; married; white; Russian; painter.

HISTORY. *Present Illness.*—On January 12, 1918, the patient came to the clinic complaining of stiffness in both lower extremities, tiredness on walking, and occasional falling because of "stubbing the toes."

The condition began in August, 1917, with stiffness and tiredness of both legs, was gradual in onset, and progressive in character. It advanced slowly but perceptibly, and until recently the patient had no pain. Since December, 1917, he occasionally experienced an aching feeling in the knees. He had no bladder or rectal disturbances. Since 1916 he has gradually become impotent. He never had headaches, nor did he vomit. Appetite is good and bowels are regular. Urinates three or four times by day and once at night. Sleep is normal. The patient thinks he lost some weight. He claims that since August, 1917, he has seen double on reading without glasses. His vision is good for distant objects and the glasses overcome the double (?) vision. On January 26, 1918, the patient stated that for the past three or four days he felt a stiffness in both upper extremities.

Previous History.—His previous history is practically negative. He had malaria in 1880; thinks he had "asthma" several years ago, and complained of occasional cramps in the legs in 1913. In 1916 a bolt fell on his head; he was dazed for a while but was not unconscious, had no bleeding from nose or ear and no subsequent headache. His habits always have been good. He had three cups of tea daily; did not drink; smoked moderately; bowels were always regular. Denies venereal diseases.

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* Presented at the Clinical Conference, February 18, 1918

Family History.—Father died at 82, of cancer; mother died at 68, cause unknown. Paternal and maternal grandparents died in very advanced age. Collaterals are all normal. Three brothers and one sister living and well; one brother and one sister died in childhood. Has four children living. Wife had one stillbirth, one child died at birth, and one miscarriage (induced)—all the last three pregnancies.

VOLUNTARY MOTOR SYSTEM. *Attitudes.*—Normal. Flat feet R>L.

Gait.—The gait is spastic. Cannot walk on his toes or heels or in a straight line.

Coördination.—Normal for non-equilibratory tests. There is no Romberg, but patient cannot stand on either foot alone whether with the eyes open or closed.

Skilled Acts.—These are practically normal.

Reflexes.—(0 = absent; 1 = slight; 2 = active; 3 = very active; 4 = with clonus):

(*a*) *Deep.*

	Right	*Left*
Jaw	2	2
Pectoral	2	2
Biceps	3+	3+
Triceps	3+	3+
Radial	3+	3+
Ulnar	3+	3+
Wrist	3+	3+
Suprapatellar	4+	4+ (clonus)
Patellar	4+	4+ (clonus)
Achilles	4+	4+ (clonus)
Dorsocuboidal	0	0
Dorsoflex	0	0
Periosteal (uncrossed)	2	2
Periosteal (crossed)	0	0

(*b*) *Superficial.*

Ciliospinal	1	1
Supra-umbilical	1	?
Suprapubic	1	?
Upper lateral abdominal	1	?
Lower lateral abdominal	1	?
Cremasteric	1	0
Plantar flexion	0	0
Babinski	p?	p?
Chaddock	0	?
Oppenheim	?	0
Gordon	0	0
Schäfer	0	0

Abnormal Involuntary Movements.—Slight, fine, moderate, regular tremor of hands. At first examination no fibrillary twitchings were observed. On January 26, 1918, fibrillations were noticed in deltoids. February 2, 1918, the fibrillations extended to biceps, triceps, suprascapulars, and infrascapulars of both sides.

Muscle Strength.—Somewhat diminished in both upper and lower extremities.

Muscle Status.—Hypertonia of both lower extremities. Electrical reactions not taken.

Abnormal Associated Movements. — The trunk-thigh and reënforcement signs of Babinski present in both legs.

NERVE STATUS.—Normal.

GENERAL SENSORY.—*Touch.* Touch localization and discrimination were normal.

Pain, Temperature, Vibratory Pressure, Muscle Tendon and Stereognostic Senses.—All found normal.

CRANIAL NERVES AND HEAD. *Cranial Nerves.*—These were carefully examined and found normal.

Optic and Oculomotor Apparatus.—Dr. W. Holden's report on the eyes states that the pupils, eye muscles, fundi and vision were all normal. There was no nystagmus. On January 26, 1918, the right pupil was somewhat larger than the left. Subsequent examinations showed them to be equal.

Mouth.—The mouth showed pyorrheic teeth.

Acoustic and Ear.—Hearing was normal, both air and bone conduction.

MENTAL STATUS.—Normal.

CRANIAL MORPHOLOGY.—Normal.

SYSTEMIC. *Tegumentary.*—Skin and hair normal. No abnormal pigmentation. Slight dermographia. No edema.

Glandular.—Cervical, axillary, epitrochlear, inguinal glands, the thyroid and tonsils all normal.

Cardiovascular.—Pulse 66 and regular. Arteries normal. Heart normal. Blood pressure: 120 systolic, 62 diastolic.

Respiratory.—Normal.

Gastro-intestinal.—Normal.

Genito-urinary.—Normal.

LABORATORY TESTS. *Wassermann.*—Blood and spinal fluid, negative.

Blood Count.—Blood count showed some degree of anæmia: reds 4,100,000; whites 12,000; hemoglobin 75 per cent., differential normal.

Urine.—No sugar, albumin or casts.

DIFFERENTIAL DIAGNOSIS.—In the subjoined schematic, diagnostic chart several possibilities have been considered in the differential diagnosis, with amyotrophic tying lateral spinal sclerosis. As is well known the latter condition is of rare occurrence. Erb first described it in 1875. He laid stress upon several points, though in his description of the symptom complex he admitted of great latitude. He pointed mainly to the absence of sensory changes, bladder and rectal disturbances and paralyses, and to the presence of great spasticity and hypertonicity. According to his description the associated movements are common. The abdominals and cremasteric may be diminished. The motor cerebral nerves may be spastically affected, giving tense expression to the face, difficulty in speech, chewing and swallowing, and spasm of the glottis. There may even be compulsory

	Lateral Sclerosis	Amyotrophic Lateral Sclerosis	Multiple Sclerosis	Cerebrospinal Lues Chronic Myelitis	Combined Sclerosis	Spinal Gliosis	Pseudo-sclerosis
Onset and History	10	7	6	6	5	3	10
Spastic Gait	10	10	10	10	5	10	10
Skilled Acts	10	8	3	8	6	6	8
Deep Reflexes	10	8	10	10	5	5	5
Superficial Reflexes	10	10	5	6	7	5	5
Fibrillary Twitchings	3	10	2	2	0	8	0
Abnormal Associated Movements	10	10	8	10	8	8	8
Absence of Sensory Changes	10	10	10	8	8	0	10
Absence of Eye Symptoms	10	10	3	4	10	10	10
Negative Serology	10	10	10	0	10	10	10
	93	93	67	64	64	65	76

laughing and crying. Pathologically, one finds degeneration of the pyramidal tracts. Erb admitted the possibility of the extension of this degeneration to the medulla, pons and internal capsule. As a transition to amyotrophic lateral sclerosis the degeneration may extend to the spinal motor centers, and he even found some degeneration in the columns of Goll and Burdach. Obviously, the latter are not pure cases. The condition is commonly found in the second to the fourth decade. As to etiology, Erb considers endogenous causes—familial and hereditary, and exogenous—intoxication, nutritional disturbances (anemia), trauma, postsyphilitic, lathyrism, pellagra, lead.

Strumpell agrees that the disease picture is an entity. According to him the condition may stop at any level in the cord. He recognizes five types: (1) familial, hereditary, endogenous, beginning in the second to third decade, progressive, and leaving out the upper extremities; (2) infantile type; (3) the type beginning late in life and progressing up to the bulb; (4) exogenous type; (5) postpuerperal cases. Obviously his classification confuses etiology, pathology and chronology; nevertheless he, too, recognizes the possibility of transitional cases. The same author, however, has in a recent contribution on the subject of "The So-called Pseudosclerosis of Wilson's Disease," described several cases, occurring in advanced ages, which he sought to explain on the pathological involvement of the extrapyramidal system. The latter condition gives a symptom complex which he would call amyostatic, in contradistinction to cases of muscular rigidity due to involvement of the pyramidal tract—myodynamic. All cases of spastic rigidity, according to him, should be investigated from the standpoint of extrapyramidal involvement, even though they do not show tremors, psychic symptoms, mimic and fixation rigidity.

The case under consideration presents nearly all of the symptoms postulated by Erb and Strumpell and, considering the possibility of transitional cases, the fibrillary twitchings observed need not deter us from making the diagnosis. Amyotrophic lateral sclerosis, however, is the equal competitor, and it may well be that we deal with such a case, although it usually does not begin in the way this case did, is not infrequently unilateral at first, and the muscular atrophy which most commonly begins in the hands often precedes the spastic paresis. I think we may consider our case as one of amyotrophic lateral sclerosis.

Final Diagnosis. *Descriptive.*—Amyotrophic lateral sclerosis.

Etiological.—Lead (?).

Anatomical.—Spinal pyramidal tracts and anterior cervical horn.

DISCUSSION

Dr. I. J. Sands.—Judging from the chart, this appears to be a case of lateral sclerosis. There is a question as to bulbar involvement in some of these cases, and I think this is one of them.

Dr. Randal Hoyt.—Before ruling out the diagnosis of a comparatively common disease and accepting one that is extremely rare, one should be absolutely sure of the accuracy of the physical examination. In this case I do not feel at all certain that there is not an atrophy of the trapezii; they certainly are proportionately small in comparison with the general musculature of the patient. The diminutive size of these muscles and the established fibrillary twitchings of the upper extremity suggest a destructive lesion in the anterior horns of the third and fourth segments of the cervical cord and an irritative lesion in the lower segment.

The involvement of both pyramidal tracts is, of course, obvious, so that the pathological requirements of a diagnosis of amyotrophic lateral sclerosis are present. The one unusual feature of this case is the fact that the lesion occurs in the upper cervical region rather than the lower. I am, however, inclined to believe that this conception of the localization of the lesion in amyotrophic lateral sclerosis is an erroneous deduction based upon habitually unsystematic physical examinations.

Dr. Frank M. Hallock.—I saw this patient when he first came to the clinic, and since Dr. Hoyt has called attention to the possible atrophy of the shoulder muscles, I remember getting the same impression the first time I saw him. The fibrillations were also present at that time.

CHOREA FOLLOWED BY PARALYSIS, SIMULATING ANTERIOR POLIOMYELITIS*

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The following is a case of unusual interest in my opinion because it illustrates a mistake in diagnosis which was made after a comparatively thorough neurological examination. When I first saw the patient I was awake to the need for a careful differential diagnosis, and although the patient came to me only on two occasions, I felt reasonably certain that my conclusions regarding her were correct. Recently however I learned of subsequent developments which make me equally certain that my interpretation of the pathological findings was incorrect. Being impressed with the fact that some of the most instructive experiences we have are those in which we make mistakes, I have asked the patient to come here this afternoon for presentation.

Patient: H. T.; Case No. 10,106; age 16; female; single; white; born in United States; schoolgirl.

History. *Present Illness, beginning with Complaint.*—The patient came to the Clinic on March 19, 1917, with the complaint that her right hand was numb and powerless. The duration was about nine weeks; the course of the disease was stationary; the onset was acute.

Previous Illness.—She had always been well until a year previous to her admission, when she was sent home from school because her teacher said that she was too excitable and twitchy. Dr. John H. Nolan tells me that he treated her over a period of about eight weeks for chorea, involving chiefly the right side of the body, including the face, but extending somewhat to the left side. She was kept in bed until all twitching disappeared. She was given Fowler's solution in increasing doses, until forty-five drops were taken daily, and then in correspondingly decreasing doses. All medication was stopped four or five months previous to admission here.

The patient states that two months ago (January, 1917) she awoke one morning to find that her right hand was powerless, and that she could not brush her hair. There were no constitutional symptoms whatever. The patient felt well in every other respect, and had been perfectly well since the onset of her paralysis. The condition has remained identically the same to the date of admission.

Her menstrual periods, previously regular, had been occurring sometimes twice in four weeks during the past year. There was no particular discomfort at the time. The systemic condition was otherwise entirely negative.

Family History.—There was nothing of any importance in the family history.

PHYSICAL EXAMINATION

Voluntary Motor System. *Attitudes.*—There were no abnormal attitudes noted, except that of marked wrist-drop on the right, when the patient attempted to use the hand.

Gait.—The gait was normal.

Coördination.—Coördination tests of the equilibratory and non-equilibratory types were normal (excluding the paralysis in performing tests).

Skilled Acts.—Skilled acts were normally performed.

Reflexes.—(1 = Slight)

(*a*) *Deep.*—	*Right*	*Left*
Supinator	1	1
Biceps	1	1
Triceps	1	1
Patellar	1	1
Achilles	1	1
(*b*) *Superficial.*—		
Epigastric	1	1
Abdominal	1	1
Plantar	1	1

Abnormal Involuntary Movements.—After a very careful investigation for a prolonged period on two occasions, no involuntary movements of any kind could be discovered. Particular caution was taken to observe the presence or absence of fibrillary twitchings or irregular movements in all parts of the body.

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* Presented at the Clinical Conference, March 11, 1918

Muscle Strength.—Muscle strength throughout was normal, with the exception of that observed in the right forearm and hand. There was marked wrist-drop, with no power whatever in the extensors of the wrist. The fingers could not be extended. The wrist flexors were normal, and flexion of the fingers was normal. The interossei and lumbricales were definitely very weak. The power of the triceps and biceps was normal and equal to that of the left side.

Muscle Status.—The muscle status was normal throughout the body, except in the right forearm, where very slight generalized atrophy was observed. The consistency and contour were normal. No abnormality of tone could be determined. The following is a transcription of notes made upon the electrical reactions:

"Muscle of right forearm and hand respond to faradism promptly. No change of formula with galvanism, excepting extensors of wrist; faradic loss, equality of anodal and cathodal closures."

Abnormal Associated Movements.—There were no abnormal associated movements.

NERVE STATUS.—The nerve status was normal.

GENERAL SENSORY EXAMINATION. *Touch, Pain, Temperature.*—Tests for touch, pain and temperature were entirely negative throughout. On two occasions exhaustive tests were made upon the right upper extremity and no abnormality was found.

Vibration, Pressure, Muscle-tendon, Stereognostic and Barognostic Sensation.—Not tested.

CRANIAL NERVES AND HEAD. *Olfactory and Nose.*—The sense of smell was not tested.

Optic and Oculomotor Apparatus.—Vision was not tested. The pupils were equal, regular, central and reacted to light, accommodation and consensual tests. There was no strabismus, no ptosis and no nystagmus. The fundi were negative.

Trigeminal and Mouth.—No abnormality could be found in the trigeminal distribution.

Facial and Face.—The facial nerve was normal on both sides.

Acoustic and Ears.—These tests were not made.

Glossopharyngeus, Vagus, Pharynx and Larynx.—The ninth and tenth cranial nerves were normal.

Spinal Accessory.—There were no abnormalities.

Hypoglossus and Tongue.—Nothing abnormal was observed.

MENTAL STATUS.—The mental status was normal. The patient was not emotional or excitable during examination. Nothing was observed that could indicate a neurotic make-up.

SYSTEMIC EXAMINATION.—The tegumentary and glandular status was normal. Special note was made of the absence of vasomotor change in the right upper extremity. The heart, lungs and viscera were normal. Systolic blood pressure was 110.

LABORATORY TESTS.—I regret to state that, as I saw the patient only twice, no opportunity was offered for making laboratory tests.

SUMMARY AND DIFFERENTIAL DIAGNOSIS

Before taking up the consideration of differential diagnosis, it may be well to summarize the pathological findings. We are dealing with a paralysis of acute onset, involving not only the extensors of the wrist but also the lumbricales and interossei. There is no sensory disturbance whatsoever. There is moderate atrophy of the forearm, and this does not seem to involve any particular muscles. The electrical reactions indicate an organic lesion of the lower motor neurone type. The course has been stationary over a period of ten weeks with no progression or regression at any time.

In the light of later developments, I think it may be well to consider the questions which suggested themselves at first.

Peripheral neuritis is usually of slow onset, of a progressive course, bilateral and symmetrical in its distribution. Sensory disturbances also usually correspond to the areas of motor involvement. Arsenic was considered as a possible factor in the production of this manifestation, but as no arsenic was administered within four months of the appearance of the paralysis, and as the symptom was unilateral, we felt that peripheral neuritis could be ruled out.

Pressure paralysis was ruled out, because there was no history of undue pressure being exerted upon the nerve trunks and because in this case I felt certain that there was involvement of two peripheral nerves—namely, the posterior interosseous as well as the median (digital branches). Furthermore, without sensory disturbance, pressure paralysis never occurs.

Hysteria was seriously considered as a productive factor. A most careful investigation of the patient's make-up, together with detailed inquiry into the patient's home life did not furnish sufficient groundwork for the establishment of a functional paralysis. It is true that the patient, because of her chorea, had been removed from home and placed in the care of an aunt. This was done because the home life was not congenial and she could not obtain sufficient rest in that environment. No reason could be found for the possible projection of an incapacitating wrist-drop into the patient's daily life. Bearing in mind that there was no sensory loss and that the motor weakness seemed to involve two peripheral nerves, I was convinced that hysteria could be eliminated as a productive factor.

The absence of tremor and fundus changes, together with normal superficial and deep reflexes ruled out multiple sclerosis.

Anterior poliomyelitis was next considered as a possible etiological factor in the production of this unusual picture. The sudden onset of the paralysis with moderate atrophy and a definite deviation from the normal in the electrical reactions strongly suggested the diagnosis. I knew of no lower motor neurone lesion which might select two peripheral nerves such as the posterior interosseous and the digital branches of the median without sensory involvement, except anterior poliomyelitis; therefore the diagnosis was established. Treatment was subsequently prescribed, but unfortunately, I did not see the patient in the clinic again.

SUMMARY

In the introduction of this article I stated that a mistake had been made in diagnosis, and my reason for so believing is as follows:

A few weeks ago I learned from Dr. Nolan that the patient had made a sudden recovery, following an operation at Bellevue. I was able to locate the patient and to obtain the following history. On June 29, 1917, she had an attack of acute appendicitis and was admitted to Bellevue with a temperature, said to have been extremely high. She was operated upon immediately and remained at the Hospital for a few weeks. Two or three weeks after her return home, she observed for the first time that she had perfect power in the right hand, and she has maintained it ever since. Examination yesterday revealed absolutely normal power in all muscles of the forearm and hand.

Analysis of the patient's history to date, therefore, shows that the paralysis developed at least five or six months after the gross choreic manifestations had disappeared. In this interval there may have been involuntary movements, but they were not sufficiently prominent to disturb the patient. The paralysis lasted at least six months and then disappeared in what one might consider a very short interval of time.

We know, of course, that paralysis, especially of the hemiplegic type, is not rare in chorea. Paralysis may precede irregular movements, be coincident with, or follow them. But I know of no case having been reported which showed paralysis which involved one or more peripheral nerves, appearing several months after the gross manifestations had disappeared and lasting for six months, with comparatively sudden disappearance.

However, as this paralysis developed in a girl who had suffered for several months from chorea, even though she might have been considered cured from the clinical standpoint, I believe it had its origin in the underlying choreic disturbance. I believe that my original diagnosis was incorrect, and although I fully appreciate the fact that the whole picture is a most unusual one, I believe that this girl's paralysis was an episode occurring in chorea.

DISCUSSION

Dr. Frederick Tilney.—This is an interesting case in many ways, but I am not sure that Dr. McKendree's error in the first place was an error, or that his second conclusion was the correction of an error. I have yet to see the case where an isolated nerve zone was picked out in chorea as in this instance. I can hardly believe that the possibilities of an hysterical condition have been sufficiently gone over to eliminate that in the differential diagnosis, and I should lean strongly to that supposition. I do not doubt that we find in chorea many cases where an extensive paralysis does exist but it is usually of the flaccid type. One thing might be suggested to make sure we are not dealing with one of those transitory palsies that occur in specific neuritis, a spinal fluid and blood Wassermann. I think it would be pertinent to this question to have this test made. We know that fugitive palsies do occur in this disease. Personally, however, I should lean to the diagnosis of hysterical palsy in this case.

Dr. William M. Leszynsky (by invitation).—If I understood Dr. McKendree correctly, the electrical examination showed a loss of faradic irritability in the paralyzed extensor group. We do not expect to find loss of faradic irritability in muscles paralyzed from hysteria. On the other hand, if the correctness of the statement can be substantiated beyond doubt, that the patient *suddenly* recovered during the time she was in this condition, that would of course indicate the psychogenic character of the paralysis.

Furthermore, if it can be shown that the patient recovered gradually although rapidly, and that faradic reaction was abolished in the paralyzed muscles, then from the history one must conclude that in all probability there was a mild focal poliomyelitis. Poliomyelitis limited to one group or part of a group of muscles is uncommon but not rare. I recently saw a child who had passed through the recent epidemic, in whom the paralysis was limited to the adductor and flexor brevis pollicis of one hand. The child is rapidly recovering the use of the affected muscles.

Dr. John H. Nolan.—When I first saw this patient two years ago, I diagnosed the case as chorea on the ground of the twitching and involuntary muscular movements of one side of the body and face. But I remember distinctly now that she had some reason for wishing conditions in her home were happier; there was dissension between her father and mother and the patient was trying to adjust matters. I remember that sometimes she laughed in a rather curious way. After eight weeks of treatment for the choreiform movements she seemed to be all right and I left her alone.

Some time before she came here to the Clinic, I met her and referred her to Dr. McKendree for dropped wrist. He told me he thought the case an atypical one of poliomyelitis and this diagnosis satisfied me. Eight weeks ago I happened to meet her again and she said her wrist was all right. She had been operated upon for appendicitis and shortly afterward the wrist was as strong as ever. I found out then that she and her mother had left the father and were living contentedly together. I have seen chorea mollis in children precede, accompany and follow this disease but never so late as in this case. It seems to me this patient's condition was probably a hysterical chorea from the very beginning. My opinion, therefore, is that her paralysis was also a manifestation of hysteria.

Dr. Louis Aronson.—I cannot see that the diagnosis of hysteria explains such physical findings as the electrical changes, or the muscular atrophies. Such signs are more likely to be associated with choreic paresis. The sudden disappearance of the paralysis is in favor of hysteria, but the question arises as to whether the patient's statement is correct. She had hyperpyrexia and was too ill to know, and without corroboration her observation was not reliable. It might have been weeks before power returned to her wrist.

Dr. M. Grossman.—The occurrence of hemiplegia in chorea is not frequent. I have had a few cases of chorea mollis under observation, one of which I reported a few years ago, a girl subject to recurrent attacks of chorea over a period of eight or nine years. She married and after becoming pregnant had a severe choreic attack lasting several months, gradually getting worse as the pregnancy advanced. Her husband left her and soon after she developed paraplegia which was accompanied by the disappearance of all choreic manifestations. She remained paralyzed for twelve to thirteen months during which time all forms of medicinal therapy were tried without avail. The only objective physical finding was a slight bilateral atrophy of the thighs and legs which I attributed to disuse. Electrical reactions, sensations and reflexes were normal. I decided that the manifestation was not paralysis that followed chorea but mainly that of an hysterical nature, and on that basis in twelve psychomotor reëducational treatments she was again able to walk perfectly well. Several other cases that I have observed showed a marked functional element.

The case under discussion today seems to me to be more or less separated from a choreic element. There was a definite period of time elapsing between the choreic movements and the appearance of the palsy. In the cases I have seen the tendency was to involve the entire limb and not pick out one group of muscles, and the paralysis appeared during the active choreic period or very shortly after the twitchings stopped. I am inclined to believe this palsy is a hysterical manifestation; the sudden disappearance of the wrist-drop would tend to make one strongly suspect it is of that nature. The electrical reaction is hard to explain, however, on a functional basis. A slight amount of atrophy does sometimes occur in cases of functional paralysis, but very infrequently.

Dr. Charles A. McKendree.—Several of the gentlemen who have discussed this case seem quite certain that hysteria was the basis of the patient's paralysis. As I have previously stated, this possibility was seriously considered and ruled out by the facts in the case. The patient was primarily choreic and her irregular twitching movements lasted over a period of several months. Mention has been made of the fact that the home surroundings were not congenial and that the patient may have developed hysterical paralysis in order to obtain for herself something that she did not have. This conception is erroneous because the patient had already been taken to the home of an aunt in order to treat her chorea successfully. The development of a wrist-drop did not become an asset to her. Of course, her chorea may have been psychogenic in origin, but if the paralytic phenomenon was projected as the expression of an unfulfilled wish I, personally, cannot understand or believe it.

One of the speakers emphasized the fact that the girl had been somewhat emotional during her illness. Who of us is not familiar with irritability and a generally disturbed mental state during the course of chorea? I do not consider this patient of a hysterical type; neither does she appear to me to represent the neuropathic make-up that one usually discovers in individuals who have suffered from chorea.

It has been said here that in chorea, paralyses are never seen in which isolated nerves are picked out. I have never seen such a phenomenon myself, previously. But, on the other hand, has anyone here ever seen hysteria select isolated peripheral nerves without sensory disturbance? I have tried to emphasize the fact that she had not only extensor paralysis of the wrist, but also marked weakness in abduction and adduction of the fingers.

Naturally the question arises, how can one account for the abnormal electrical responses? That I cannot answer except to say that there is always a possibility of error, especially when the reaction of degeneration is not complete and deviates from the normal only slightly.

With regard to the sudden recovery of the patient; I have been unable to ascertain exactly when this phenomenon took place. She was very ill for two weeks following the operation and the nurse did everything for her. Later she told the nurse that she had a paralysis of the wrist and the nurse continued to wait upon her, making it unnecessary for the patient to use the hand. The hand was not normal when she returned home, but her aunt tells me that a few weeks later power was entirely restored. My interpretation of the comparatively sudden return of power is not that of psychic shock, but that of reaction following a severe toxemia.

SUPERIOR ALTERNATING HEMIPLEGIA—WEBER'S SYNDROME

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and
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The two cases presented in this report have interest in the fact that they illustrate a syndrome due to entirely different pathological disturbances occurring in patients widely separated in point of age. The simultaneous appearance of these two cases in the wards of the Presbyterian Hospital has afforded unusual opportunity for a comparative clinical study of Weber's syndrome.

Superior alternating hemiplegia or Weber's syndrome consists of a complete or partial paralysis of the third cranial nerve on one side, causing ptosis, external strabismus and internal ophthalmoplegia combined with a hemiplegia involving the face, arm, trunk, and leg of the opposite side of the body. Anatomically the lesion is situated in the mid-brain, especially involving the emerging fibers of the oculomotor nerve and the cerebral peduncle.

The first exact account of this syndrome, both clinically and anatomically was given by Weber in 1863. It had been clinically described with much precision of observation by Mohr in 1840 and by Green in 1852. Gubler in 1859 gave a description of a case of alternating hemiplegia in which there was a paralysis of the right oculomotor nerve and a left hemiplegia. This he found to be due to softening of the right cerebral peduncle. Upon Charcot's proposal in 1891 this combination of symptoms, due to lesion affecting the cerebral peduncle, has been designated *Weber's Syndrome*.

Case I.—Patient: E. B.; female, Italian; age 3. (Case Histories No. 20229 V. C. and No. 39555 P. H.) Admitted to the Vanderbilt Clinic, January 23, 1918.

History. *Present Illness.*—One night in October, 1917, the patient was put to bed in good physical condition. In the morning the mother found her paralysed. The paralysis involved the right side of the body, but the mother observed at the same time that the child could not raise her left eyelid. She was also very irritable and did not talk as usual. She had no fever, vomiting, convulsions, or loss of consciousness. She had no difficulty in swallowing. Her condition has been stationary since the onset.

Previous History.—She is the third child of apparently healthy parents, born March 30, 1915, after an easy labor. She had no cyanosis, fever, or convulsions during her first month. She was nursed for thirteen months. She held her head up well at four months, sat up at eight months, and walked at ten months. She cut her first tooth at one year and also spoke at this time. At two years she could speak short sentences in the Italian language.

She had measles at one year, whooping cough at one and a half years but has had no known exposure to tuberculosis.

When she was two years and five months of age, she fell out of bed and fractured her left elbow. She recovered in a few weeks and had no further illness until the present one.

Family History.—The father and mother are alive and well. There is no history of miscarriages, still births, or abortions. The three other children are in good health: There is no evidence of any familial nervous disorders.

Voluntary Motor System. *Attitudes.*—No abnormal attitudes were observed.

Gait.—The baby is unable to stand or walk.

Coördination.—There is no coöperation.

Skilled Acts.—There is no coöperation.

Reflexes: (0 = absent, 1 = slight, 2 = active, 3 = very active, 4 = with transient clonus, 5 = with permanent clonus):

Deep:	*Right*	*Left*
Biceps	3	2
Triceps	3	2
Radial	2	1
Patellar	4	2
Achilles	5	1
Periosteal	4	3
Crossed periosteal	1	1
Superficial:		
Abdominal	0	1
Epigastric	0	1
Plantar	0	0
Babinski	1	0

Abnormal Involuntary Movements.—There are no involuntary movements.

Muscle Strength.—There is diminished muscle strength in the right upper and lower extremity, otherwise the motor tests were normal.

Muscle Status.—The child's muscles are generally soft and flabby, more marked on the right side of the body.

Electrical Reactions.—The electrical reactions were omitted.

Abnormal Associated Movements. — These tests were not tried.

GENERAL SENSORY.—The general sensory examination was omitted as the child was too young to respond.

CRANIAL NERVES AND HEAD. *The Olfactory Nerve and Nose.*—These tests were omitted.

The Optic Nerve and Oculomotor Apparatus.—The child apparently sees its mother and cries when strangers approach. The fundi are normal. The pupils are unequal, the right measuring 2 mm., the left 5 mm. The right pupil is slightly irregular, the left pupil is circular; both pupils are concentrically placed.

The right pupil reacts normally to light. The left pupil did not react at all. The accommodation tests cannot be elicited. There is no consensual reaction. There is a marked ptosis of the upper left eyelid and external strabismus in the left eye.

The Trigeminal Nerve and Mouth.—The sensory and motor division cannot be tested with the exception of the corneal reflexes which are present and equal.

Facial Nerve and Face.—There is a central type of paralysis of the right side of the face. The left facial nerve is normal.

Acoustic Nerve and Ear.—Hearing tests cannot be made due to lack of coöperation, but the child's hearing appears normal.

Glossopharyngeus and Vagus Nerves; Pharynx and Larynx.—The glossopharyngeus and vagus nerves, the pharynx and larynx give no evidence of abnormalities.

Spinal Accessory and Hypoglossal Nerves are apparently normal. Motion of the tongue is normal, although it is impossible to test protrusion. There is no atrophy of tongue.

SYSTEMIC EXAMINATION. *Tegumentary* status is normal.

Glandular Status.—There are no cervical, axillary, epitrochlear, or inguinal lymph nodes palpable at the first examination.

The child was admitted to the Presbyterian Hospital in March, 1918. While under observation there, a small tumor developed in the left axilla. It was excised for diagnostic purposes. The pathological report was: Tuberculosis of an axillary lymph node.

While in the hospital, her temperature ranged from 97.2 to 100. The pulse varied from 100 to 130. The respirations were 20 to 28 per minute.

The *Cardiovascular, Respiratory, Gastro-intestinal and Genito-urinary* systems are normal.

Skeletal Status.—There was a slight deformity around the left elbow and a small swelling over the right malar bone.

LABORATORY TESTS.—The blood Wassermann of the child was negative on three occasions. The mother's serum was also negative.

The spinal fluid showed:

cells 20
globulin 4+
Wassermann negative to 2 c.c.
colloidal gold 55555555+

The Von Pirquet reaction was negative the first time. The second test was 1+ after 12 hours and 2+ after 24 hours and negative after 48 hours. The third test gave the same reaction as the second.

The blood examination showed: Hemoglobin 78%; red cells 2,500,000; white cells 6,200, of which 76% were polynuclears and 24% lymphocytes.

The urine was clear. The specific gravity varied from 1022 to 1025. Reaction was acid. There was no albumen or sugar.

The *x*-ray of the left elbow and right malar bone showed no distinct abnormalities. Other *x*-ray pictures of the skull and general osseous system were normal. The thymus was slightly enlarged.

SUMMARY AND DIFFERENTIAL DIAGNOSIS.—This case presents a right-sided hemiplegia involving the face, and also a complete third nerve palsy of the left side. The symptoms developed over night and have not progressed. Clinically the symptom-complex is that of Weber's syndrome.

A word might be added with reference to the marked irritability of the child, and also to the speech which was formerly spontaneous and is now retarded. I believe these manifestations are due to the intracranial disorder.

While the clinical diagnosis of superior alternating hemiplegia is readily made from the symptoms, the etiology of the disease is open to debate. It seems desirable therefore to chart the various possibilities and give each symptom its percentage according to the neurological findings.

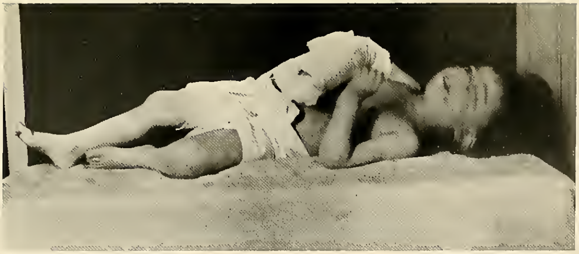
The diagnosis which obtained the largest percentage is tuberculous meningo-encephalitis or solitary tubercle involving the left crus cerebri. The following tabulation gives the reasons for this diagnosis.

CASE II.—Patient: W. K.; male, colored, laborer; age 33. (Case No. 37155 P. H.) Admitted to the hospital, January 22, 1918.

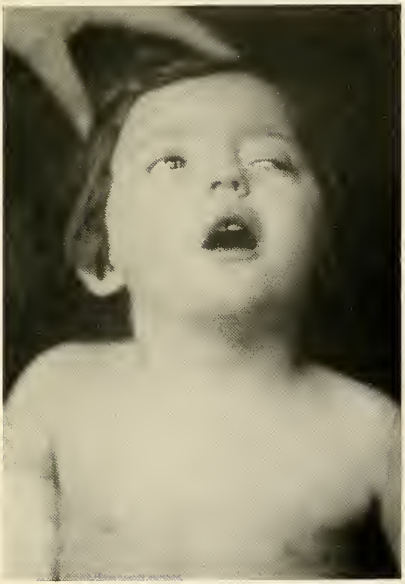
HISTORY. *Present Illness.*—In October, 1917, the patient noticed that the upper lid of his right eye began to droop and that he frequently saw double. In the course of three weeks, the upper lid drooped to such a degree that the right eye was entirely closed and at the same time the right eyeball became permanently turned outward. In the early part of January, 1918, he observed that it was increasingly difficult to move his left arm and leg until at length he had quite lost the use of his left hand, while his gait became slower due to weakness and dragging of the left leg.

Five months ago (September, 1917) he began to feel faint and dizzy for periods of twenty or thirty minutes at a time, such attacks occurring on the average of four to six times a day. This disturbance was aggravated by stooping over and closing his eyes. The dizziness has improved slightly. The weakness in the left arm and leg of which he complains came on suddenly, for one morning on attempting to rise from his bed, he fell. He found that it was impossible for him to stand or walk but he did manage to drag him-

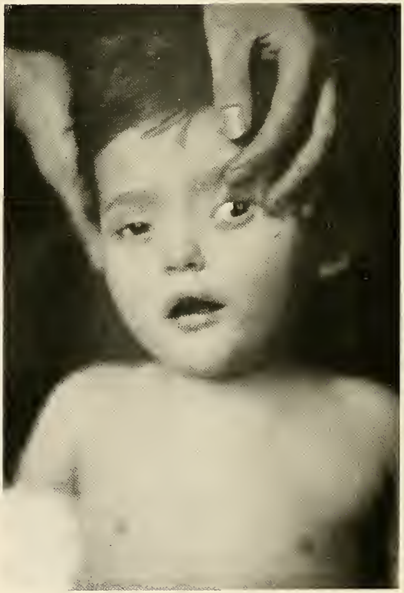
	Tuberculous Meningo-encephalitis or Solitary Tubercles	*Polien-cephalo-myelitis*	*Encephalitis (Influengol-pyogenic)*	*Tumor*	*Gumma or Meningo-encephalitis*	*Hemorrhage Thrombosis Embolism*	*Abscess*
History	5	10	2	2	2	2	0
Onset	10	10	4	5	3	3	3
Course	10	5	2	3	10	10	3
Fundi normal	8	10	5	0	5	8	3
Absent—Convulsions or loss of consciousness	10	10	2	0	10	0	0
Absent—Vomiting	10	10	5	0	10	5	0
Absent—Fever	10	10	0	10	10	2	3
General condition	6	8	3	2	6	5	0
Laboratory	10	10	5	3	0	0	0
Biopsy	10	0	0	0	0	0	0
	89	83	28	25	56	35	12



Showing Alternating Paralysis



Showing Upper Facial Intact



Showing External Strabismus

self to the sink. Here he found that he could not use his left hand to turn on the water. Since this attack the paralysis has remained but little changed. He thinks there may be some improvement in his leg but believes his arm has become worse. He has pain and tenderness in his right ear. This has been constant since October, 1917, although never severe enough to disturb his sleep.

He is married and has one child now six years of age. His wife had no miscarriages. Until one year ago he drank heavily. He eats and sleeps well. His best weight was 201 pounds but in the past four months he has lost thirty pounds.

According to his previous history he has enjoyed unusually good health. He has had no acute infectious diseases. Although he has been cut several times in fights, he has sustained no serious injuries. In February, 1918, he was in Bellevue suffering from a sore throat which was thought to be syphilitic in origin. Two years ago he had a cold and expectorated bright red blood in small quantities. Since then he has coughed a good deal but there has been no more blood. For the past few months he has had night sweats sufficient to dampen the bed clothes. During the past five years he has had nocturia. He never had gonorrhea but eleven years ago contracted syphilis for which he received more or less desultory treatment. He never suffered from frequent or severe headaches, convulsions, jaundice, nervous or mental disorders, impairment of sight or hearing.

The family history shows that his father died at seventy years of age of consumption. His mother and three sisters are alive and well. One sister has heart disease. Otherwise the family history is negative.

VOLUNTARY MOTOR SYSTEM. *Abnormal Attitudes and Deformities.*—There were no abnormal attitudes or deformities.

Gait.—Left hemiplegia with swinging of the left leg in the phase of progression.

Coördination.—All movements of the right arm and leg are normally performed. Nonequilibratory coördination is normal. In the left arm and left leg the paralysis makes it impossible to estimate what degree, if any, of ataxia is present. All tests for equilibratory coördination were normally performed.

Reflexes (0 = absent, 1 = slight, 2 = active, 3 = very active):

Deep:	*Right*	*Left*
Jaw	0	0
Pectoral	1	1
Biceps	1	4*
Triceps	1	4*
Radial	1	4*
Ulnar	1	4*
Wrist	1	5†
Suprapatellar	0	3
Patellar	2	5†
Achilles	2	5†
Dorsocuboidal	1	1
Periosteal	0	0
Superficial:		
Ciliospinal	1	0
Supra-umbilical	2	1
Suprapubic	2	1
Upper lateral abdomen	2	1
Lower lateral abdomen	2	1
Cremasteric	0	0
Plantar	0	0
Babinski	0	2
Chaddock	0	2
Oppenheim	0	2
Gordon	0	0
Schäfer	0	0

Abnormal Involuntary Movements.—There were no tremors, twitchings, spasms, athetoses, convulsions, or choreiform movements reported or observed.

Muscle Strength.—Marked loss of strength in entire left side of body. The right upper and lower extremities showed no loss of muscle strength.

Muscle Status.—There was no change of contour, volume, or consistence in the right upper and lower extremities. The left arm and leg are smaller than the corresponding parts on the right side. The muscles upon the left side of the body are slightly hypertonic.

The electrical reactions show no change in the normal contraction formula in the muscles and nerves on the right and left sides of the body. There are no quantitative changes in the electrical reactions on either side.

Muscle irritability is normal upon the right and left sides.

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* With clonus. † With permanent clonus.

Abnormal Associated Movements.—The observations concerning these pathological associations in motor activity are of such interest in every case of hemiplegia as to warrant more extensive attention than they usually receive. For this reason the following tabulation is given in this case.

	Right	*Left*
1. Pronation sign of Strümpell	o	Active
2. Radialis sign of Strümpell	o	Active
3. Automatic pronation sign of Babinski	o	Active
4. Bechterew's forearm sign	o	Active
5. Mechanical grip sign	o	o
6. Thumb sign of Klippel-Weil	o	Active
7. Interosseous sign of Souques	o	o
8. Raimiste's sign of flaccidity	o	o
9. Heilbronner's sign of flaccidity	o	o
10. Supination sign of Neri	o	Active
11. Genuflexion sign of Neri	o	Active
12. Trunk-thigh sign of Babinski	o	Active
13. Reenforcement sign of Babinski	o	Active
14. Leg elevation sign of Grasset-Byschowski	o	Active
15. Complementary opposition sign of Grasset	o	Active
16. Abduction-adduction sign of Raimiste	o	Active
17. Tibialis sign of Strümpell	o	o
18. Sign of Marie-Foix	o	Active
19. Coughing sign of Huntington	o	Active
20. Contralateral associated movements	o	Active
21. Identical associated movements	o	o

In connection with the abnormal associated movements, it is of interest to note the relatively large number of those phenomena present in a case of comparatively slight upper motor neurone involvement.

General Sensory. *Touch acuity*, localization, and discrimination are normal upon each side of the body.

Temperature (including cool, warm and hot), vibratory pressure and muscle tendon sensibility is also normal upon the right and left sides of the body. Pain stimuli due to pin point, to pinching the skin as well as skin and muscle, pulling the hair, sharp percussion over subcutaneous periosteal surfaces and over extension at all of the joints gave a less definite response upon the entire left side than upon the right. While no algometric tests were employed it seems fair to take the patient's statement that painful stimuli felt twice as strong upon the right side of the body and gave him the sensation as if the left side of his body was "numb to the pin-prick except the left cheek" where pin point is sharp. Electrical stimulation by means of the faradic current showed thresholds for current perception in selected areas of the body as follows:

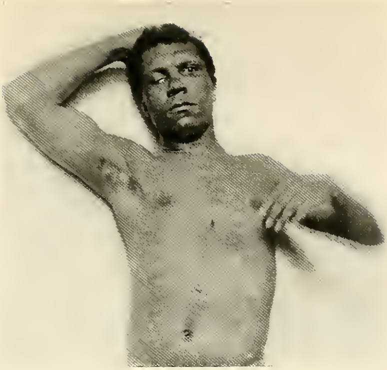
Area	*R. Coil Distance*	*L.*
Face	3.00 cm.	3.00 cm.
Neck	3.00 cm.	3.25 cm.
Arm	4.00 cm.	4.00 cm.
Forearm	3.75 cm.	3.50 cm.
Hand	4.00 cm.	3.50 cm.
Fingers	3.00 cm.	3.50 cm.
Trunk	3–4.50 cm.	4.25 cm.
Thigh	5.00 cm.	4.50 cm.
Leg	4.50 cm.	5.50 cm.
Foot	4.25 cm.	5.00 cm.
Toes	4.25 cm.	5.00 cm.

Stereognosis and *barognosis* are normal.

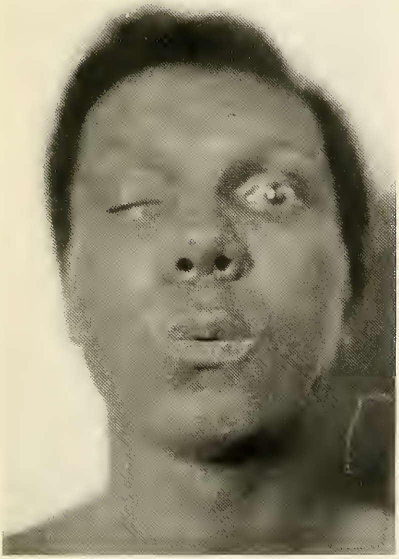
Cranial Nerves. 1. *Olfactory nerve and nose* are normal.

2. *Optic nerve*, visual apparatus, oculomotor nerves, and oculomotor apparatus. The vision is fair if objects are held well to the temporal side of the right eye, otherwise vision becomes indistinct. Perimetry shows no scotomata but a marked contraction to the visual fields upon the left side in upper quadrants, *i.e.*, an upper quadrant left homonymous hemianopsia. (See chart.) The eyes show a slight pallor of both discs. The right pupil is larger than the left, the former appearing to be somewhat dilated. The light reflexes are normal in the left eye but absent in the right. The accommodation reflex shows a similar defect in the right eye. There is no enophthalmos or exophthalmos in either eye. A marked, complete ptosis is present in the upper right eyelid, while the right eye assumes an external strasbismic position. There is no spontaneous nystagmus in any position of the eyeballs. The winking and blinking reflexes are normal in both eyelids and associated as is usually the case in health.

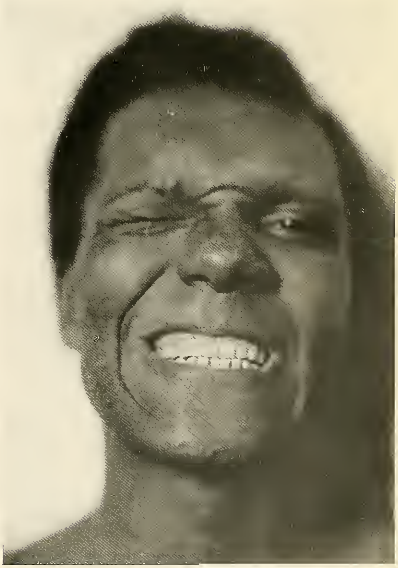
To summarize the optic and oculomotor disturbances, it may be stated that there is a complete internal and external right ophthalmoplegia with the exception of involvement of the abducens and trochlearis nerves. The visual disturbance is a left homonymous hemianopsia affecting the upper quadrants of the visual fields.



Showing Alternating Paralysis



Showing Facial Palsy



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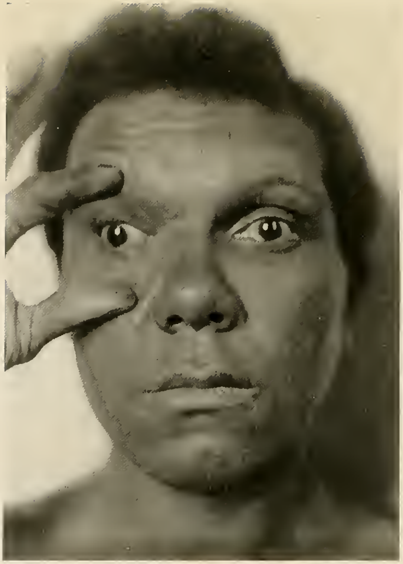
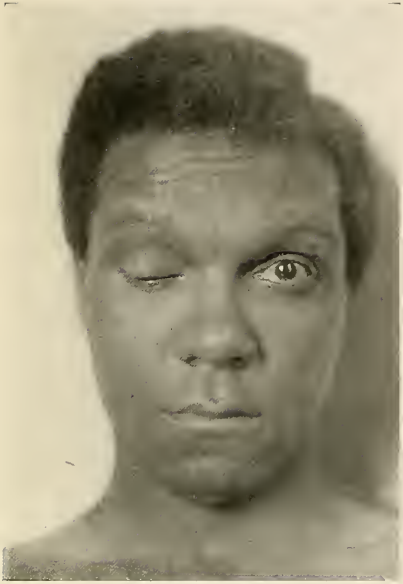
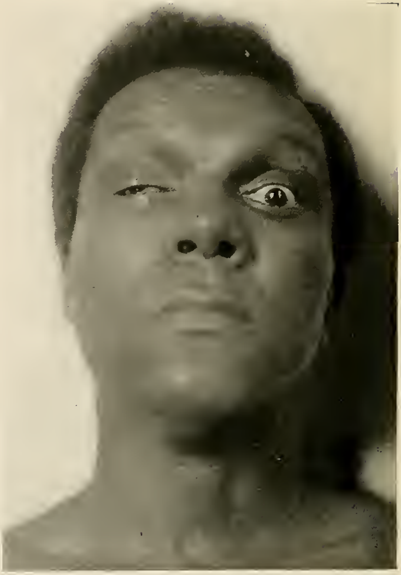


Showing External Strabismus



Showing Facial Palsy

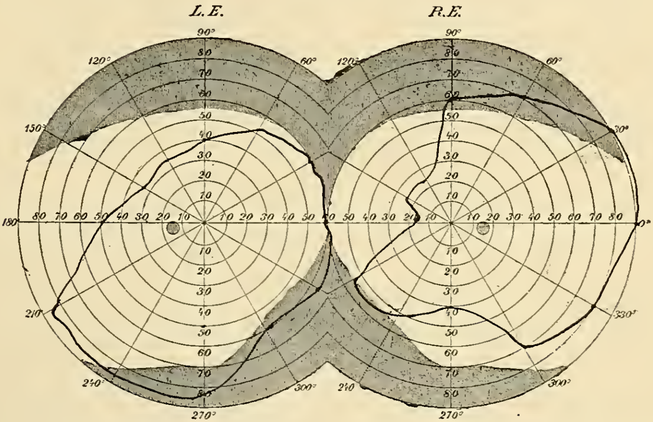


Showing Upper Facial Intact

The Acoustic Nerve and Ear.—(*a*) Cochlear division of the eighth nerve is normal. There are no subjective disturbances and all objective tests yielded normal response, *i.e.*, Rinné and Weber.

The vestibular division of the eighth nerve gave the following responses to the Bárány and Hitzig tests.

Bárány: Cold water in right ear caused a lateral nystagmus (horizonto-rotatory) with slow movement to the right after 25 seconds. Nystagmus persisted for 130 seconds. Both eyes participated in the slow movement to the right, but the right eye shows very little of the rapid movement to the left so conspicuous in the left eye. The test in this ear causes much more vertigo than in the left ear and the patient complained of some nausea. There was no vomiting. Cold water in left ear in 30 seconds produced a nystagmus similar to that due to stimulation of right ear. The movements, however, lasted only 70 seconds and were unattended by vertigo or nausea. The Hitzig test showed a normal response to 6 amp. of current from anode and cathode applied to either ear.



Trigeminal Nerve and Mouth.—There is no paralysis in the muscles of the jaw. The conjunctival and orbicular reflexes are present. Pin point seems sharper on the left side of the face. This is noteworthy since the left side of the body was generally less sensitive to the pin point than the right side.

Facial Nerve and Face.—The motor control of the right side of the face was normal in voluntary and emotive expression. The left side of the face showed a conspicuous palsy in the lower and middle portions, while the upper facial muscles showed a normal innervation. There was no change in gustatory sensation on either side of the tongue in its anterior two-thirds.

Glossopharyngeus and Vagus Nerves, Larynx and Pharynx.—The sensory and motor functions of these two nerves were normal, and showed no alterations in secretion, position of uvula, and vocal cords, phonation, swallowing respiration, or cardiac action.

Spinal Accessory Nerve and Neck were normal.

Hypoglossal Nerve and Tongue showed no departure from the normal.

Mental Status was normal in all respects.

Systemic Examination.—Revealed no abnormalities in pigmentation, sweat secretion, or vasomotor status.

The temperature ranged from $98^{2}/_{5}$ to 101 during his confinement to the hospital. During this time he received several injections of salvarsan which were followed by a slight rise in temperature. There were some enlarged lymph nodes in the postcervical group.

Pulse ranged from 64 to 84; the blood pressure was systolic 120, diastolic 75.

Condition of the arteries and heart was normal.

The respiratory, gastro-intestinal and genito-urinary systems were normal.

LABORATORY TESTS

Spinal fluid:

Amount removed 12 c.c.

Pressure slightly increased

Cells	310			
Globulin	2 +			
Wassermann	2 c.c.	1 c.c.	.6 c.c.	.2 c.c.
Alcoholic antigen	0	0	0	0
Cholestrin antigen	3+	2+	0	0

Blood Serum Wassermann

Alcoholic antigen	4+
Cholestrin antigen	2+

Blood Count

Hemiglobin	84%
Red cells	4970000
White cells	7900
Poly	74%
L.L.	12%
S.L.	12%
Trans.	2%

Urine analysis, normal except for a few finely granular casts seen upon one examination.

Roentgenogram of skull and tibia shows periosteal thickening of both tibiae; the sella turcica is small but the skull furnishes no evidence of increased intracranial pressure.

DISCUSSION OF CASE II

There would seem, perhaps, little ground for the discussion of the second case, since the neurological findings clearly define the condition as Weber's syndrome while the serology shows the disturbance to be due to neurosyphilis. On the other hand, it is of more than passing interest to decide, if possible, which of the several forms of neurosyphilis this may be. Such a decision should be arrived at not only for the sake of exactness in diagnosis but quite as much in its bearing upon the prognosis.

Further than indicating the presence of a syphilitic process the laboratory tests have little significance. The colloidal gold curve would assist but little in the differential diagnosis since the possibility of the paretic type of syphilis has not been raised in connection with the case. The diagnostic problem hence is to distinguish between diffuse neurosyphilis, vascular neurosyphilis, meningitic neurosyphilis, encephalic gummatosis, or gumma. Much too often the significance of clinical observation is underrated in favor of laboratory findings, but in this case, as frequently happens, it is the clinical findings which furnish the most valuable, indeed, the only real aid in the differential diagnosis. Perhaps, it is not speaking extravagantly to maintain that anatomy still plays the dominant role in neurological diagnosis.

There are two symptoms which assume much significance in this connection, *i. e.*—

1. Partial left homonymous hemianopsia.
2. Hypalgesia and hypothermo-esthesia upon the left side of the body.

Both of these are relatively uncommon complications of Weber's syndrome. The hypalgesia of the left side increases in importance when taken in conjunction with the fact that all other qualities of sensibility are normal upon the left side of the body even to the more delicate electric sensory tests.

What is the significance of the partial left homonymous hemianopsia?

Accepting the general midbrain level of the lesion it becomes evident that the visual defect must be due to an involvement of the optic fibers in or near their primary receiving center in the *lateral geniculate body.* The partial character of the hemianopsia indicates that not all of the visual fibers have been affected, hence the lesion is not likely to be one due to compression, for in that case the hemianopsia would tend to be complete.

The hypalgesia and hypothermo-esthesia of the left side of the body with the absence of any changes in the other qualities of somatesthetic sensibility speaks in favor of a lesion within the brain substance rather than one compressing it. It is quite possible that an encephalic lesion in the midbrain might involve the spinothalamic tract (pain-temperature) without affecting the mesial fillet; in fact, the spinothalamic tract, sweeping upward from the midbrain into the interbrain, would come into relation with the lateral geniculate body before the mesial fillet. Of necessity such a lesion would be in the substance of the brain for it is impossible to conceive of compression in the region so selective as to affect the spinothalamic tract and not the mesial fillet. An encephalic lesion in this light, must be accepted as more probably the patholgical process rather than a ectencephalic involvement causing compression. For this reason gumma and syphilitic meningitis may be ruled out.

Vascular neurosyphilis does not seem to satisfy the situation because the distribution of vessels in this region is too diffuse to conceive of a strictly vascular disturbance as the cause of the disorder. On the other hand, although the lesion may not be a compact one, it is definitely focal and localized in the midbrain levels. This would seem to eliminate the possibility of a diffuse neurosyphilis. The only other possibility is meningo-encephalic gummatosis, that is a more or less scattered infiltration of the meninges in the region of the emergence of the third nerve and the pyramidal tract in the right cerebral peduncle. This infiltration would also involve the tegmentum in the region of the spinothalamic tract as well as the lateral geniculate body.

The completed diagnosis offered in the case, therefore, is Weber's syndrome due to meningo-encephalic gummatosis.

The prognosis is good and the patient, with proper intensive antiluetic treatment, should make a complete recovery. Already several intravenous treatments have been given and a marked improvement is apparent in the symptoms.

THALAMIC SYNDROME WITH LEFT HEMIANOPSIA AND RIGHT OPTIC ATROPHY*

By OLIVER S. STRONG, Ph.D.
Assistant Professor of Neurology, Columbia University
NEW YORK CITY

Patient: M. W.; No. 20,109; age 31 years; female; married. After leaving school at sixteen years of age, she worked in a carpet mill for a number of years, was idle a year or more, and from then until her marriage at the age of twenty-six worked in a cigarette factory.

HISTORY. *Present Illness beginning with Complaint.*—The patient's principal complaint is a gradual loss of sight toward the left. In December, 1916, she began to notice a clouding in the left side of the right eye. She consulted Dr. Knapp, who advised her to have her teeth treated by a dentist. The patient did not follow his advice at the time, but the teeth were removed a month or so ago. In six months the cloudiness increased and the left eye began to be involved, at first the very end of the left side and gradually spreading toward the middle. At present she can see objects on the right but not on the left. These visual disturbances vary at different times. She has worn glasses since she was twenty years of age.

She also complains of being easily fatigued —especially in the feet and body, and of a trembling—especially in the right arm and hand, but some also on the left. This last symptom is referred back even before the time of the accident, mentioned below, which occurred six years ago and which the patient thinks made it worse. After this accident she was "paralyzed in her legs" and could not walk for three weeks.

While pregnant with her first child, now four years old, the patient in lifting her foot high on a toilet seat "ripped her stomach," which was sore for a long time. A month after this she lost sight, apparently in the left eye. This lasted three months. At the same time she had a pain in the left side of the head and lost hearing on the left side. The onset was sudden. This lasted also three months until the birth of the child, when blindness, deafness and pain in the head, according to the patient, suddenly and entirely disappeared. Labor was painless.

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* Presented at the Clinical Conference, February 18, 1918

She also complains of pains in the back which sometimes awaken her at night but are better than a year ago. She used to get numb in the hands a year ago; this has gone now.

For "going on three months" she has suffered from "cramps in the stomach" and since early in January has been troubled with headache. One day (January 16) she had "an awful pain in the head," attributed by her to her stomach. She is troubled with flatulence and attributes this to tea drinking. She perspires very profusely in summer.

Previous History.—No definite information is obtainable concerning her early development. As far as she knows she thinks it was normal. She was taken care of for a time by the Gerry Society.

There is no history of injuries to the head. The patient was hit with a packing case across the thighs some six years ago; this is the accident referred to above. She had measles when small, malaria when fifteen or sixteen years old, and whooping cough a year or so ago. At one time she had some hoarseness—"could not talk." There is no history of convulsions nor of paralysis, except after the accident, the weakness of the hand being called nervousness. Her menstruation is usually regular, rather small in amount. (January 9, none for five weeks; February 11, three months pregnant.) She has been married five years and has had two children, both living and said to be healthy. No miscarriages. There is no incontinence of urine; she urinates five to eight times a day and once or twice during the night, this latter being attributed to tea drinking. She does not use coffee, alcohol, tobacco or drugs, but drinks about six cups of tea a day. Her appetite has been poor lately. She has always been constipated. There has been apparently no significant change in weight.

Family History.—There are not many details obtainable. Her father died at fifty-six years of age. He had malaria and rheumatism and was "very nervous." Her mother died at forty-six of kidney trouble. She was not nervous, was "healthy," but was an alcoholic. It was apparently the latter fact that led to the patient's being in charge of the Gerry Society.

VOLUNTARY MOTOR SYSTEM. *Abnormal Attitudes and Deformities.*—There is some tendency to kyphosis.

Gait.—The gait has not been thoroughly studied, but seems fairly good considering the patient's general condition. She walks well on her toes; not so well on her heels.

Coördination.—Standing with eyes open, the patient shows perhaps slight but hardly significant unsteadiness. There is no significant increase with the eyes closed. She cannot stand on one foot with eyes open or closed.

When performing the finger-to-nose test slowly, eyes closed, the hand as it approaches the nose gradually sinks and she touches below the nose. The test is much better performed rapidly. At times there is a tremor of the intention type, but disappearing when the test is rapidly performed. The tremor is apparently increased by fatigue.

In the heel-to-knee test, eyes closed, the patient at first always touched the right heel to the left thigh some distance above the knee. At a subsequent visit she did not do this, but on both sides moved the foot up and down in a rather tentative way before touching the knee. Apparently there is no adiadochokinesis in the arms but the movements are rather slow, and the patient says she soon gets "played out" doing them.

Skilled Acts.—Speech seemed normal. Tests showed possibly a slight dysphasia.

Reflexes.—(0 = absent, 1 = slight, 2 = active, 3 = very active, 4 = with transient clonus, 5 = with permanent clonus):

(*a*) *Deep.*—

	Right	*Left*		*Right*	*Left*
Biceps	2+	2	Patellar	3	2+
Triceps	4	2			
Radial	4	2+	Supra-patellar	3	2
Ulnar	4	2−	Achilles	3	2
Wrist	3	2+	Periosteal (shin)	1 [?]	0

The clonus on percussing the radial is rotatory (*i. e.*, pronation and supination).

It is doubtful whether it may not be permanent rather than transient. The apparent periosteal is not the true adductor reflex but an over-reaction to the percussion.

(*b*) *Superficial.*—The abdominal reflexes are active and there is an over-reaction, especially on the right. The plantar is present and there is no Babinski nor any of its modifications, but in testing the plantar there is more general reaction on the right and the patient said "it went through her leg." While making the Chaddock, Oppenheim, Gordon and Schäfer tests the patient complained of pain or disagreeable sensations on both sides—but more on the right.

Abnormal Involuntary Movements.—There is at times, at least, a tremor in the right hand and arm. This tremor, which is apparently both motofacient and non-motofacient, seems to be in part a fatigue tremor associated with the easy fatigability of the part involved and in part clonic in character. It does not appear to be an intention tremor. It is partly rotatory.

Muscle Strength.—The pareses found are not very marked, and further examination would be desirable to make sure of their correct identification. Pareses of the following appeared to be present: Abduction and adduction of the right arm; extension of the right (and left?) forearm; adduction of the right fingers; flexion of the right (and left?) thigh, possibly also abduction and adduction; extension of the right and left legs; flexion of the right leg; and dorsal flexion of the right foot. The tests appeared to demonstrate, in some cases, the fatigability complained of by the patient.

Abnormal Associated Movements.—These were not tested but it was noticed that the sign of Grasset-Bychowski (inability of the recumbent patient to elevate both legs at once) was present.

NERVE STATUS.—Not tested.

GENERAL SENSORY.—This has not been exhaustively examined, but the following peculiarities were observed:

Touch.—Touch discrimination was tested on the second finger tips, forearms and dorsal surface of the feet. The finger tips were found to be normal (2 to 3 mm. right and left), the anterior surface of the right forearm showed a diminution as compared with the left (3½ cm. and 1½ cm.), and the right foot showed a diminution as compared with the left (3 cm. and 2 cm.). Subsequent tests on the forearm did not show this difference so clearly.

Pain and Temperature.—It seemed doubtful that there were any objective changes in pain and temperature.

Vibratory.—Vibratory sense seemed absent in the right shin and apparently diminished in other parts of the right side.

Muscle-tendon.—The sense of position and movement seemed normal in the big toes and feet, and possibly so in fingers, hands and arms. There were at times, however, phenomena such as those seen in the finger-to-nose test, which indicate that a more rigid testing might reveal some abnormalities here, especially perhaps of the sense of position.

When making the above tests, however, peculiarities of reaction became evident which might be characterized as a hyperesthesia, or better as an over-reaction or an increase in affect. This was shown more markedly, perhaps entirely, on the right side. The pain tests (needle point) were much more disagreeable to the patient on the right side. This peculiarity was not shown equally over the whole right side, being slight and possibly not present on the back and face. Moderately cold and hot stimuli showed to some extent the same reaction. Light touch when repeated on the same area, so as to tickle, showed the same phenomenon; *e. g.*, the right side of the face would twitch and the patient said it "went through her more" apparently "like a cold chill." This could not be demonstrated on the fingers. In all three, pain, temperature and touch, the patient usually said that equal stimuli on the two sides were equal; *i. e.*, she thought she was touched with equal force on the two sides. Sometimes she said the stimuli were duller on the right. Percussion of the shins, chest, ulnars and forehead gave more reaction ("irritated more") on the right. On the other hand, percussion of the dorsum of the feet and pressure on the legs seemed to give an over-reaction on both sides.

CRANIAL NERVES AND HEAD. *Olfactory.*—Not tested.

Optic Nerve and Oculomotor Apparatus.—Dr. Holden (January 28) found the following: Pupils: 5 mm., round. Light reaction: good. Convergence reaction: good. Hippus. No diplopia. Nystagmus: slight to left. Mobility: normal. Vision with her myopic glasses: Rv = movements of hand; Lv = 20/100. Fields: complete, absolute, left homonymous hemianopsia. Right eye: movements of hand are seen in the temporal half of the field, but a 1 cm. test object is not seen. Vision in right eye varies from day to day. Left eye: nasal half of field is complete for 3 mm. test object. Fundi: conus to nasal side of each disc. Right disc is diffusely pale; left disc of normal color. A left homonymous hemianopsia due to lesion behind basal ganglion. Right atrophy of optic nerve: peripheral. Right progressive loss of vision followed by left homonymous hemianopsia.

The fields were plotted by Dr. Schoenberg (January 18). The left eye (for white, 10 mm. object) shows a loss of the left (temporal) half just to the median line and a slight contraction of the right (nasal) half. The right eye (finger movement) shows a loss of the left (nasal) half extending beyond the median line and a further considerable contraction of the right (temporal) half.

Trigeminus.—One observer found the corneal reflex was nearly absent on the right and diminished on the left. This was not confirmed by Dr. Holden.

Facial and Face.—Upper and lower facial movements were normal excepting possibly at times a slightly greater contraction on the right in certain movements. There was some tremor on both sides during contraction. Taste was probably normal (sweet, normal; bitter, ?) on right and left.

Acoustic and Ear.—Hearing appeared to be normal on both sides, though one observer thought there was some diminution on the left.

Glossopharyngeus, Vagus, Pharynx and Larynx.—The pharyngeal reflex was present. Taste was normal.

Hypoglossus and Tongue.—The position of the tongue when protruded was normal or possibly slightly to the right. The movements of the tongue were normal.

MENTAL STATUS.—This was not carefully examined. The patient said she thought her memory was poor: "she forgot where she put things." There seems to be some emotional deterioration. She is rather apathetic.

CRANIAL MORPHOLOGY.—Nothing noted.

SYSTEMIC.—Not thoroughly examined.

Tegumentary.—Varicose veins were noted on the left leg and a small subcutaneous lump in the small of the back on the left side near the median line.

Cardiovascular.—Blood pressure was 120 systolic, 85 diastolic. The heart was normal.

Gastro-intestinal.—Gastric flatulence has been noted. The abdomen was distended.

LABORATORY TESTS. *Wassermann.*—Two Wassermann tests made at the Knapp Hospital were negative.

Lumbar Puncture.—There was an unsuccessful attempt at lumbar puncture thwarted by a "hysterical condition," really probably due to the peculiar sensory status of the patient.

X-ray.—An undated *x*-ray report from the Knapp Hospital is as follows: "Sella turcica is contracted but is clearly outlined. No tumor visible. The sphenoid is clear. No evidence of bone absorption. The skull seems to be very thick. No dilation of meningeal vessels. No pressure there. Sella turcica 10 mm. long, 4 mm. deep."

SUMMARY

The salient results of the physical examination are, in brief:

1. Active deep reflexes, very active on the right with clonus in the upper extremity.
2. Slight pareses, principally or entirely on the right, in the upper and lower extremities.
3. Slight diminution of objective sensibility on the right, possibly confined to tactile discrimination and vibratory.
4. Increase in affective tone of pain, temperature, tactile and pressure stimuli, most marked on the right but possibly present also on the left.
5. Left homonymous hemianopsia and right optic atrophy, the former of apparently sudden onset but gradual and varying in subsequent development and intensity, and the initial attack accompanied by left deafness and headache.

1 and 2 point to a slight involvement of the left pyramidal system above the decussation.

3 and 4 are the type of sensory disturbances seen in thalamic lesions. The principal alternative possibility would be the hyperesthesias and paresthesias seen in peripheral neuritis. These, however, are true hyperesthesias which might nevertheless be confused with the thalamic type. The one-sided character of the disturbance and their association with pyramidal symptoms (increased deep reflexes) and optic changes, together with absence of trophic changes would practically exclude this possibility. The thalamic lesion would be entirely or principally on the left side. 5 might be theoretically explained by a lesion of the right optic tract (or the right external geniculate or above the latter) and also extending far enough downward to involve other portions of the right optic nerve. The association with deafness in the first attack would point to a lesion in the region of the geniculate.

When we attempt to explain the symptoms by a single lesion we encounter the fact that the eye symptoms are due to a right-sided lesion, more probably at least two lesions (*see* Discussion), and the remaining symptoms to a left lesion. It will be noted also that the latter group, though not so striking, apparently were noticed some time before the visual symptoms attracted the attention of the patient. We seem then to be driven to the assumption of more than one or even two lesions, one, or two, of them on the right, involving the right optic tract (or geniculate or higher) and the right optic nerve, and the other in the left optic thalamus.

According to Head's conception of thalamic function, as far as sensory changes are concerned, we may get (1) a complete or partial loss of sensibility due to the interruption of the spinothalamic and fillet systems just before or at their entrance into their terminal nuclei in the thalamus; (2) an interference with that part of the path (thalamocortical) which is sent directly to the cortex and which conveys there those stimuli which are at the basis of cortical associations and memories (judgments as to space, position, etc.); (3) interference with the corticothalamic fibers which inhibit the specific thalamic activities. As these latter activities are concerned chiefly with the affective side of various stimuli, especially pain, temperature and visceral, such interference would increase the affective or emotional reactions to such stimuli. The fibers involved in (2) and (3) constitute the thalamic radiations which would therefore appear to be partly affected in the present case, the thalamic lesion consequently probably being in the lateral part of the thalamus. It is not proposed to discuss this conception at present, but it may be remarked that its anatomical basis is at present weak on the efferent side and, further, whatever the part played by the thalamus, many of the reactions forming the basis of the clinical picture, including, of course, all the language reactions, are reactions involving the cortex.

Regarding the nature of the lesions there would appear to be the following possibilities: (1) neoplasm; (2) vascular lesions; (3) multiple sclerosis; (4) cerebro spinal lues; (5) tabes. Of these, the first has against it the bilateral and apparent multiple character of the lesion, the absence of papilledema, of marked headache (which is also against thalamic lesions) and the negative x-ray. Tabes also seems improbable; none of the symptoms are *characteristic* of tabes. The negative Wassermann, mobile pupils and history are against lues. There are again few *characteristic* symptoms of multiple sclerosis except the pyramidal symptoms. Low blood pressure, and especially absence of retinal arteriosclerosis, militate against vascular lesions.

Two peculiarities that deserve notice are the sudden onset of the first eye (and ear) symptoms and their variability as to intensity (which, even discounting the patient's account somewhat, seems to me to be a real feature), being apparently increased by pregnancy. The sudden onset speaks rather for a vascular lesion, but the variability decidedly for multiple sclerosis, which *may* have a sudden onset. It is also difficult to account for the location of the lesions on a vascular basis. It seems to me, therefore, that the probabilities rather favor the disease process being of the nature of a multiple sclerosis, but of a different distribution from the more usual forms. Next in probability would come cerebrospinal lues and vascular lesions with a neoplasm less probable than the above.

DISCUSSION

Dr. Ward A. Holden.—It is unusual to find in one patient visual disturbances due to lesions of the two different sets of visual neurones. In this case, there is complete, absolute, left homonymous hemianopsia, there being not even perception of light in the left half of the field of each eye. This, of course, might mean a lesion in the optic radiation or in the optic tract. The latter is extremely rare, however, and excepting in cases of aneurysm an optic tract hemianopsia is accompanied by well-marked basilar symptoms and, after a few weeks duration, by pallor of both optic discs. The left optic disc in this case is normal in color and the right half of the field is normal for even small test objects. Furthermore, no basilar symptoms are present, and hence we must conclude that we have to do with a left homonymous hemianopsia due to a destructive lesion of the right optic radiation.

In the right eye, however, the disc is diffusely pale, vision is reduced to counting fingers at a few feet, and there is marked concentric contraction of the right half of the field for even very large test objects. Here, then, we have an optic nerve atrophy due to destruction of the visual fibers anterior to the chiasm.

The history given by the patient is confusing. She says that a number of years ago she lost the sight of the left eye (patients with left hemianopsia usually think they have lost the sight of the left eye) and later, she thinks, the vision improved. This alleged improvement is very improbable.

The visual diagnosis here must be a progressive loss of sight in the right eye, due to an atrophy of the optic nerve, preceded or accompanied by a rapid loss of vision in the left half of each visual field, due to destruction of the second set of visual neurones which extend from the thalamic region to the calcarine fissure in the occipital lobe.

Dr. M. J. Schoenberg.—Homonymous hemianopsia with conservation of the pupillary reaction is usually regarded as due to a lesion located in the cortical visual area or between that and the optic basal ganglia. But there are other possibilities which occur to me. One is that in spite of the presence of the pupillary reaction in this patient, the lesion may be in front of the geniculate ganglion. The patient has a right optic atrophy and a left homonymous hemianopsia, and the lesion which would cause these two conditions would be at the right extremity of the chiasm, involving the right optic nerve and the nerve fibers supplying the nasal portion of the retina of the left eye. The persistence of the pupillary reflex could be explained by the fact that in some exceptional cases of blind eyes and optic atrophy, the nerve fibers conveying the pupillary reflex do not degenerate and the pupils react to light in spite of the fact that the eyes are completely blind.

Another point to be considered is the character of the hemianopsia and the complaint of the patient. Hemianopsia due to a cortical or subcortical lesion is usually characterized by the presence of an irregular line dividing the seeing from the blind field. There is a bulging of the seeing field into the blind one, a so-called "*überschüssiges Gesichtsfeld*," as described by Wilbrand, in these supragangliar lesions, while in optic tract lesions the line separating the seeing from the blind field is straight with a slight deviation around the macula. In this patient we do not find that characteristic *überschüssiges Gesichtsfeld* which speaks rather against a lesion above the primary optic ganglia.

Furthermore, the patient seems to be aware of the defect in her visual field, and this again is found in tract lesions. Patients with cortical or subcortical hemianopsia are not aware of their visual field defect; they have a negative scotoma. Marie, in his study of the field of vision in soldiers with injuries of the occipital lobe, calls special attention to this character, negative scotoma, of the visual fields. The onset of a cortical or subcortical process is rather sudden and usually accompanied by grave general disturbance and bilateral blindness, from the so-called *étonnement cérébral* which later on gradually clear up. Tract lesions begin without such general disturbances. The history of this case would help to throw some light on this point.

As I have said, there are a number of points which cast some doubt upon the probability of the localization of the lesion above the primary optic ganglia. In spite of that, however, I agree with Dr. Holden's diagnosis as the most acceptable.

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DOUBLE PRIMARY ATHETOSIS

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The patient, whom I show you, presents a symptom which is sufficiently rare to be of interest, and illustrates a condition about which there is still some pathological obscurity. She has a most intense form of double athetosis.

The symptoms consist of constant irregular spontaneous movements, which are much increased by any mental excitement or by any physical exertion, and cannot be arrested by effort. They involve the face, causing a wrinkling of the eye-brows and forehead, and a slight movement of the ears; queer grimaces, and contractions of the mouth with pouting or retraction of the lips; occasional opening of the mouth and sudden spasms of the risorii muscles, all of which movements give rise to peculiar expressions of the face, like that of fear, or of anger, or of melancholy. These motions of the face are increased by any attempt to tâlk. On opening the mouth it is evident that the tongue shares in these movements, and attempts to speak are very imperfect, so that it is impossible to understand what is said by the patient [1]. The eyes are occasionally closed tight or opened unduly wide. All the extremities are also involved in these movements. There are irregular unexpected movements of the fingers and hands, and sudden throwing about of the arms, which hinder any use of the hands, such as in attempts at dressing herself and feeding herself, both of which acts are quite impossible. Her legs are also involved, so that walking is attended with great difficulty and is impossible without support, which is given by holding her up under the arms; and even the steps are irregular in length. The knees occasionally bend under her and the feet are placed in unexpected positions. There is double talipes equinus with contracture of the muscles of the calves. There is a weakness of the muscles of the neck and back and a marked kyphosis without rigidity. The muscles are well nourished; there is no atrophy and their electrical reactions are normal. The reflexes are slightly exaggerated but there is no Babinski and no clonus. Her sensibility is everywhere perfect. Sight and hearing are perfect; also smell and taste. Her mental condition is one of a mild grade of imbecility, and in talking of herself she always speaks of herself in the third person. Her mother, however, maintains that she is exceptionally intelligent, and reads to her constantly, maintaining that she understands everything that is presented to her. This case corresponds almost exactly with cases of Sterling (1), of Spiller (2), of Rothmann (3) and of Hultgren (4).

When one considers the fact that in these cases of double athetosis there is no paralysis and no disturbances of sensation, one becomes convinced that the origin of the spasmodic movements is some abnormal sensation sent upward to the cortex, which gives rise in a reflex manner to the movements produced. The movements must be of cortical origin because they have the characteristics of cortical movements, being more or less coördinated and apparently like the movements with a voluntary purpose. They are in no sense choreic in character nor are they like the movements of the various forms of tic. They differ also wholly from the movements of myoclonus, being slower and more diffuse. Nor do they have the character of the so-called rotary movements of the feebleminded, described by Weir Mitchell, which are rhythmical and which follow one another in definite order. Thus it is evident that their character is typical and cannot be mistaken for anything else.

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[1] SWIFT, W. P. Studies in Speech Disorder, No. 5—The Speech in Athetosis. *Rev. Neurol. & Psychiat.*, Edinburgh, 1916, p. 151. In this article Swift has made a careful study of the speech defect in a case of athetosis.

The association with weak-mindedness, in some cases, is accidental, for in the majority of cases recorded the mental state of the individual has been perfect. It was so in the two other cases which I have seen. One of these was a boy of seven, who presented very much the same symptoms as those in this girl. The other case was an adult male who had been similarly affected all his life.

Athetosis is a symptom which is manifested under two conditions.

First. There is a primary spontaneous general bilateral athetosis which is usually congenital or appears during the early months of life, though it has been known to develop as late as the eleventh year. It has been thought by Higier (5) to be a family disease, since he saw two children affected in the same family; but this is not confirmed by other cases. This type of athetosis is extremely rare, as is established from a study of the literature. Isolated cases have been reported from all countries, but in the past ten years there are but nine cases to be found in the *Jahresbericht für Neurologie und Psychiatrie.*

The pathology of double athetosis is still uncertain. Hammond in the ninth edition of his textbook collected thirteen cases with autopsy, in all of which the lesions found were located in or near the basal ganglia, particularly in the lenticular nucleus, though in some cases the optic thalamus was involved. Since Hammond's time Anton, Oppenheim and Vogt, Fischer, and Rothmann have recorded cases with autopsy, in each of which there have been lesions in the lenticular nuclei. Atrophy of the globus pallidus and sclerosis in the putamen are the lesions found. Such a localization of the lesions would correspond with our conclusion that the character of the movements indicates a good functional state of the cortex.

Second. Secondary athetosis or athetosis appearing in consequence of brain lesions is not at all uncommon. It is then unilateral. About ten per cent. of the children who have infantile hemiplegia develop athetoid movements subsequent to the onset of the paralysis. Athetoid movements may occur in adults as a sequel of hemiplegia. The most severe case of athetosis of a unilateral type in my experience developed subsequently to the removal of a brain tumor from the posterior central convolution in a young man of thirty and persisted for a year after the operation, but gradually subsided entirely. In these cases of secondary athetosis, consequent upon lesions of the brain, there does not appear to be any uniform location of the lesion, so that the origin of the athetosis pathologically is still a matter of doubt. Athetosis is rarely, if ever, a symptom developing in brain tumor of the cortex in the sensory or motor areas, and does not result from meningeal inflammation. In a case of unilateral athetosis with hemiplegia, operated on for me by Dr. McBurney, the meninges and cortex were apparently normal when exposed. The pathological records in literature are few and they are so diverse as not to lead to any definite conclusion as to the location of lesions producing unilateral athetosis. Subcortical cysts in the cerebrum, old cicatrices, a result of hemorrhages or softening in the thalamus, in the lenticular nucleus, in the red nucleus of the tegmentum, in the superior cerebellar peduncle, in the inferior cerebellar peduncle, and in the body of the pons, have all been found in different cases where there has been a unilateral athetosis (6). In several of Hammond's cases the symptoms were unilateral and so was the lesion.

In view of the present belief that the lesion in athetosis is located in the lenticular nucleus, it is interesting to inquire whether in Wilson's disease, which is caused by disease of this nucleus, athetosis has been observed. In the few cases I have seen there has been none.

There appears to be no treatment for this condition. Bromides and similar remedies have had no effect upon the movements. The operation performed by Spiller of division of the sensory nerve roots along the spine does not appear to have had any permanent beneficial effect.

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A CASE OF MYXOSARCOMA OF THE TEMPORAL BONE WITH EXTENSION INTO THE POSTERIOR CRANIAL FOSSA PRESENTING UNUSUAL SYMPTOMS*

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Flora J., married, 46 years of age, was admitted to the Surgical Service of Mount Sinai Hospital (Surg. Case No. 177,466) on November 12, 1917.

Previous History.—Five children, two of whom died of convulsions in infancy; three children living and well. At four years of age, as a complication of an attack of small-pox, the patient had a paralysis of the left side of the tongue, with subsequent atrophy. Soon afterward, she had a purulent discharge from the left ear followed by diminished hearing in that ear. With the exception of the atrophy of the left side of the tongue and the diminished hearing in the left ear, she was well until thirteen years ago.

At the age of thirty-three, after the birth of a child, the patient had a left hemiplegia. This gradually cleared up, excepting that a left facial weakness and a slight weakness and incoördination of the left arm persisted, and that her voice remained hoarse and her breathing somewhat stridulous. Excepting for the disturbances above described, the general health of the patient remained good until three years ago.

She then developed a large swelling in the left submaxillary region, was operated upon, and a mass of glands was removed which was said to have been of a chronic inflammatory nature. After the operation, she noticed that the left angle of her mouth "drooped" more than it had ever done before. She recovered satisfactorily from the operation and did not have any new symptoms until September 1916.

Present History.—About one and a half years ago, after a severe mental shock, the patient began to have frequent headaches and attacks of dizziness. The headaches recurred very frequently and were uninfluenced by treatment, so that six months ago she entered the New York Neurological Institute, on the service of Dr. Peterson, in order to obtain relief. At this time the paralysis of the left side of the face became complete.

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* From the Surgical Service of Mount Sinai Hospital, New York City. Presented by invitation, at the Clinical Conference of the Neurological Department of Columbia University, April 15, 1918. I am indebted to the Pathological Department of Mount Sinai Hospital for the pathological specimen and pathological report.

The following notes are taken from the records of the Neurological Institute:

"The patient's general condition is good. Her gait is normal. The pupils are unequal, the right is larger than the left (O. D. pupil 5; O. S. pupil 2.5 mm.). The pupils react normally to light and accommodation. Fundi and fields of vision are normal. The sensation of the left cornea is slightly diminished. In the extreme lateral positions, there is nystagmus, slow and coarse to the left, fine and rapid to the right."

"There is a complete left facial paralysis of the infranuclear type, and no reaction to faradism in any of the left facial muscles. To the galvanic current, there is a reaction of degeneration in the facial muscles on the left side."

"The hearing in the left ear is much impaired but is not entirely lost. Both labyrinths react normally. Dr. Dench states that the impairment of hearing in the left ear is due to an old suppuration, but that the ear is dry and the process not active."

"There is complete atrophy of the left half of the tongue. The tendon reflexes in the extremities are lively and equal; the abdominal reflexes are equal; there are no pathological reflexes in the lower extremities. Sensation is normal over the face and extremities."

"The cerebrospinal fluid obtained by lumbar puncture is clear, two cells to the cubic millimeter; globulin not increased; Fehling reaction positive; Wassermann negative."

The patient left the Neurological Institute unimproved, and her condition grew gradually worse. The headaches became more severe and were often accompanied by vomiting. Her eyesight became poor. She began to have difficulty in walking, until she was finally unable to stand or walk without assistance. She lost considerable flesh and strength. Finally, the headache became constant. In this condition, she was admitted to the Surgical Service at Mount Sinai Hospital.

Physical Examination.—The physical examination at this time (November 15, 1917) showed the following:

General condition poor. The patient is much emaciated and looks very ill. Her sensorium is clear. She speaks in a hoarse voice but in a characteristically cerebellar monotone. She is very ataxic, and falls to the left when she stands with her eyes closed and when she attempts to walk alone.

The posture of her head is normal.

Cranial nerves: The sense of smell is normal. The optic discs are hyperemic but there is no papilledema. The right pupil is larger than the left. Both pupils react to light and accommodation, the left more sluggishly than the right. In extreme lateral positions of the eyeballs, there is coarse nystagmus to the left, and fine rapid nystagmus to the right. Irritation of the sympathetic on the right side causes dilatation of the pupil, on the left, there is no reaction.

There is a hypesthesia over the entire area of the left trigeminus with anesthesia of the left cornea. The left abducens is weak. There is a complete paralysis of the left seventh. The muscles supplied by the left facial nerve do not react to the faradic current and there is marked reaction of degeneration.

There is complete deafness in the left ear with negative caloric reactions. Complete loss of function of the cochlear and vestibular divisions of the left auditory nerve (Dr. Braun). Movements of the head are good in all directions. The palate does not deviate. There is an advanced atrophy of the left half of the tongue. Both vocal cords are relaxed; the motion of the right cord is limited both in abduction and adduction, the left vocal cord is paralyzed. The voice is hoarse and the breathing sibilant.

Trunk and extremities: The abdominal reflexes are weak, and equal on the two sides. The power in the left upper extremity is weaker than in the right, and there is slight ataxia in both, but most distinct in the left.

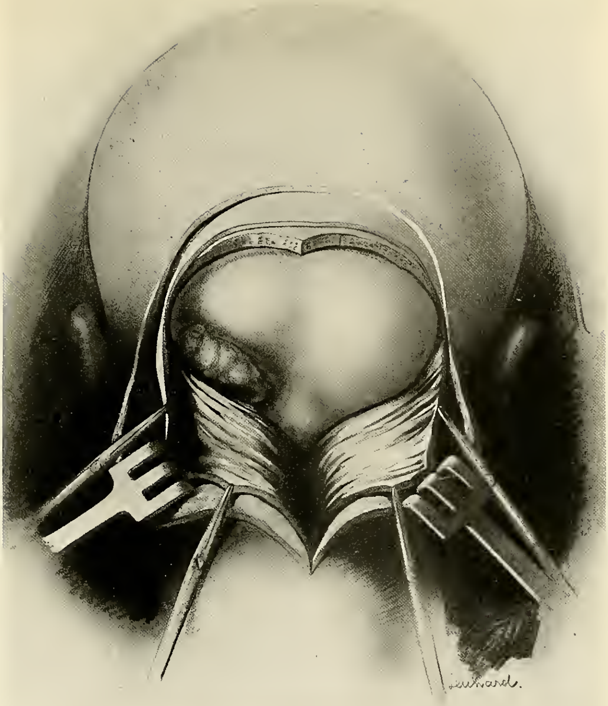
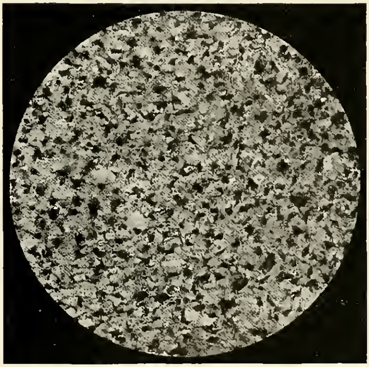


Fig. 1. The Protruding Extradural Tumor After Suboccipital Craniotomy Had Been Done



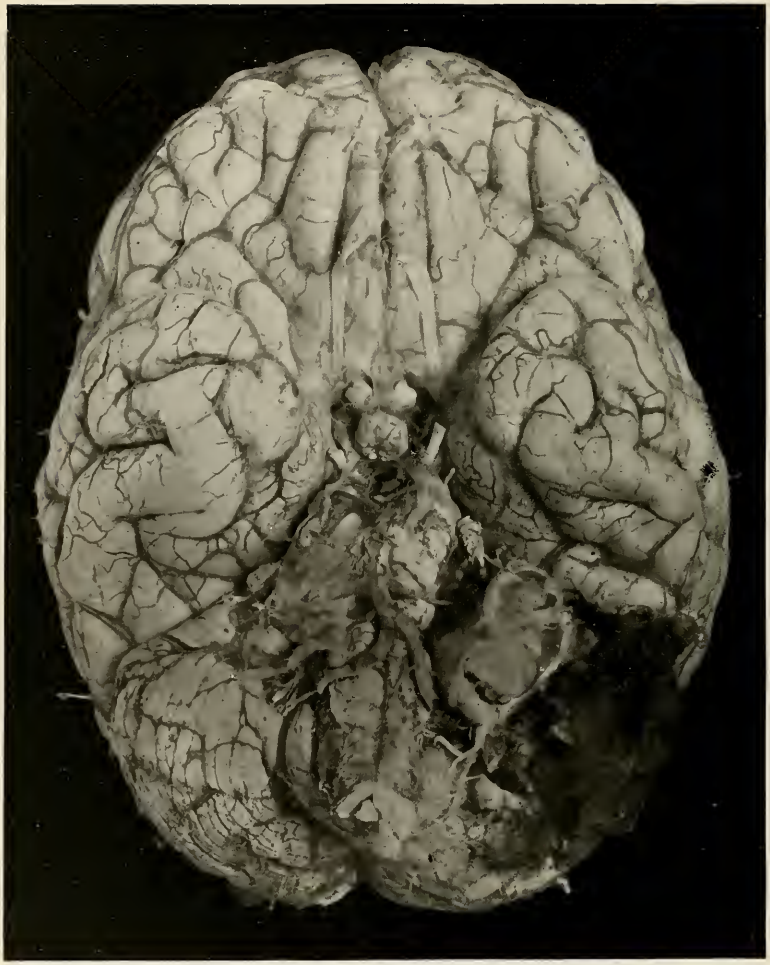


FIG. 3. MYXOSARCOMA OF THE LEFT TEMPORAL BONE INVOLVING THE POSTERIOR CRANIAL FOSSA

The photograph shows the intradural tumor mass, the outer half of which is discolored from hemorrhage into it.

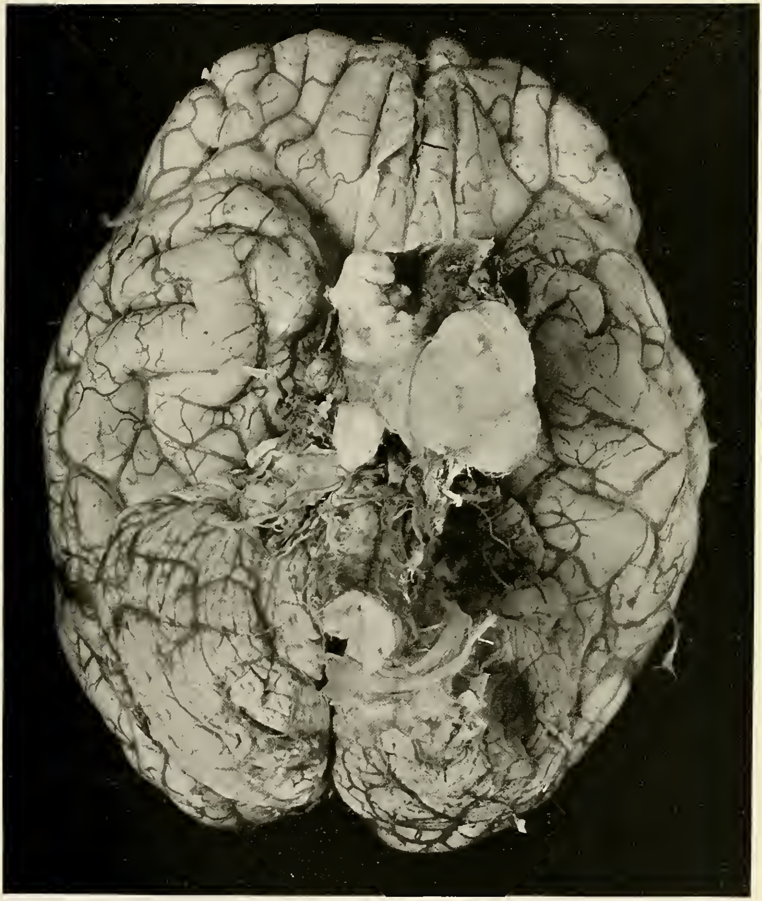


Fig. 4. The Intradural Tumor Dislocated Forward to Show the Deep Indentation on the Side of the Pons and Cerebellar Lobe

The biceps, triceps and radius jerks are more lively on the left side. There is adiadochokinesis in both upper limbs, but more marked on the left side. There is no disturbance of the superficial sensibilities, and the patient can distinguish between rough and smooth bodies on both upper extremities.

The power in the lower limbs is normal. The left lower extremity is very ataxic, by the heel-to-knee test. The reflexes are normal and equal, there are no pathological reflexes.

X-ray plates of the skull failed to show anything abnormal.

Subsequent History.—The diagnosis of a tumor in the left cerebellopontine angle was made, and the patient was operated upon (Dr. Elsberg). The dura over the cerebellum was exposed by a typical bilateral suboccipital craniotomy. As soon as the dura covering the lower part of the left hemisphere was exposed by the removal of the occipital bone, considerable yellow gelatinous material began to extrude from the left posterior fossa, and after the dura had been depressed, the left posterior fossa was found to be entirely filled by this material (Fig. 1.) Considerable of the gelatinous matter was removed with a spoon. There was some bleeding from the jelly-like substance, and it was evident that the entire side of the fossa was filled up by the mass. The wound was closed in the usual manner.

On December 14 the wound was dressed and the sutures were removed. Healing had occurred by primary union. The general condition of the patient remained poor. She died suddenly on December 15, ten days after the operation.

The pathologist reported that the material removed (*see* Fig. 2) was of the nature of a myxosarcoma (Dr. Strauss).

The post-mortem examination was performed ten hours after death (Dr. Thalhimer), but only a cranial autopsy was permitted.

The brain was of normal size and shape and the hemispheres showed nothing abnormal. There was moderate distention of the third and lateral ventricles. The right cerebellar lobe was normal, the left side of the pons and medulla and the left cerebellar lobe were deeply indented by a large tumor mass. (*See* Figs. 3 and 4.) The mass was of a gelatinous consistency, and entirely filled the posterior fossa on the left side between the dura and the bone. At one spot the growth had perforated the dura, so that a second tumor mass lay against the pons and medulla inside of the dura. Part of the tumor mass was brownish in color, evidently due to a hemorrhage into it at the time of the operation. After the brain had been removed *in toto*, the petrous portion of the left temporal bone was found to have been almost entirely destroyed by the tumor, so that only a thin shell of bone remained. The tumor had evidently originated in the petrous portion of the left temporal bone, had perforated the bone, and had filled the posterior fossa on the left side. There was a moderate foraminal depression on both lobes of the cerebellum, showing that there had been a protrusion of the cerebellum into the foramen magnum. This explained the sudden medullary death of the patient.

CONCLUSIONS

Aside from the rarity of this form of tumor in this location, the symptoms and clinical signs presented by the patient were of extraordinary interest and most confusing. In each attack of illness the left side of the body, and especially cranial nerves on the left side, were affected. The attack of small-pox followed by otitis, left behind a paralysis and atrophy of the left side of the tongue and diminished hearing of the left ear. The postpartum attack—probably one of cerebral embolism—added a paresis of the left side of the face and a weakness of the left arm with paralysis of the left vocal cord. After the removal of glands from the left side of the neck, a branch of the left facial nerve was found to be completely paralyzed (ramus marginalis mandibulae). Finally, the patient developed symptoms which were clearly referable to the left lobe of the cerebellum and the left posterior fossa—ataxia, nystagmus, adiadochokinesis, hypesthesia over the distribution of the left trigeminus, complete paralysis of the left facial, complete nerve deafness in the left ear and loss of the normal caloric reactions on that side.

It was not surprising, therefore, that the condition was at first considered to be one of extensive funicular and nuclear degeneration. A careful study made it clear that on an old nuclear lesion of the left hypoglossal nerve and an old peripheral lesion of the left eighth, that on an old partial lesion of the seventh cranial nerve, of the pyramidal tracts to the left upper extremity, and a nuclear lesion of the abductor and adductor muscles of the vocal cords, there were superimposed the symptoms and physical signs of an expanding lesion in the left side of the posterior fossa.

RUPTURED ANEURISM OF THE RIGHT MIDDLE CEREBRAL ARTERY*

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The following case of rupture of the right middle cerebral artery presents certain features of unusual diagnostic interest.

Louis J. B. was admitted to the Neurological Institute under the care of Dr. E. G. Zabriskie on December 29, 1917. The patient was a healthy-looking man, twenty-one years of age, who gave the following history:

His father died of chronic nephritis. The patient had the ordinary diseases of childhood, but otherwise he was always well. He smoked and drank moderately. Denies syphilitic or gonorrheal infection.

About December 1 the patient fell on the ice and struck his back. He says the fall jarred him considerably, but that he suffered no ill effects and was able at once to go to his business. On December 13 he went down into the cellar of his home, in order to attend to the furnace. As he stooped down to open the furnace door, he had a sudden sharp pain in his head, especially across his eyes, and felt dizzy and faint. He called to his sister, who came down to him at once and found him lying on the floor unconscious. A physician was called who found the patient in a semistuporous state, from which he could be partially aroused. This stupor lasted until the following day. When he became fully conscious, he complained of severe headache and of diplopia. He was not nauseated and did not vomit. It was noted by his physician that his neck was stiff and that he had a positive Kernig. Dr. Zingher, of Camp Upton, did a lumbar puncture upon him on that day, withdrawing twenty to thirty cubic centimeters of bloody cerebrospinal fluid. Excepting for the presence of blood, the laboratory reports on the fluid were negative; the Wassermann reaction was negative.

The patient remained in the same condition, excepting that his headaches became steadily more severe. On December 26 (thirteen days after the beginning of his illness) he had a generalized convulsion and was unconscious for one hour. Another lumbar puncture was performed, and eighty cubic centimeters of bloody cerebrospinal fluid, under considerable pressure, were withdrawn. After the second lumbar puncture the headaches became less severe. On December 29 he was admitted into the Neurological Institute, under the care of Dr. E. G. Zabriskie. At this time he complained of headache, rigidity of the neck, inability to walk, and general prostration.

December 29: Physical examination on admission. The patient is a well-nourished man.

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*From the Neurological and Surgical Departments of the New York Neurological Institute. Presented, by invitation, at the Clinical Conference of the Neurological Department of Columbia University, April 15, 1918.

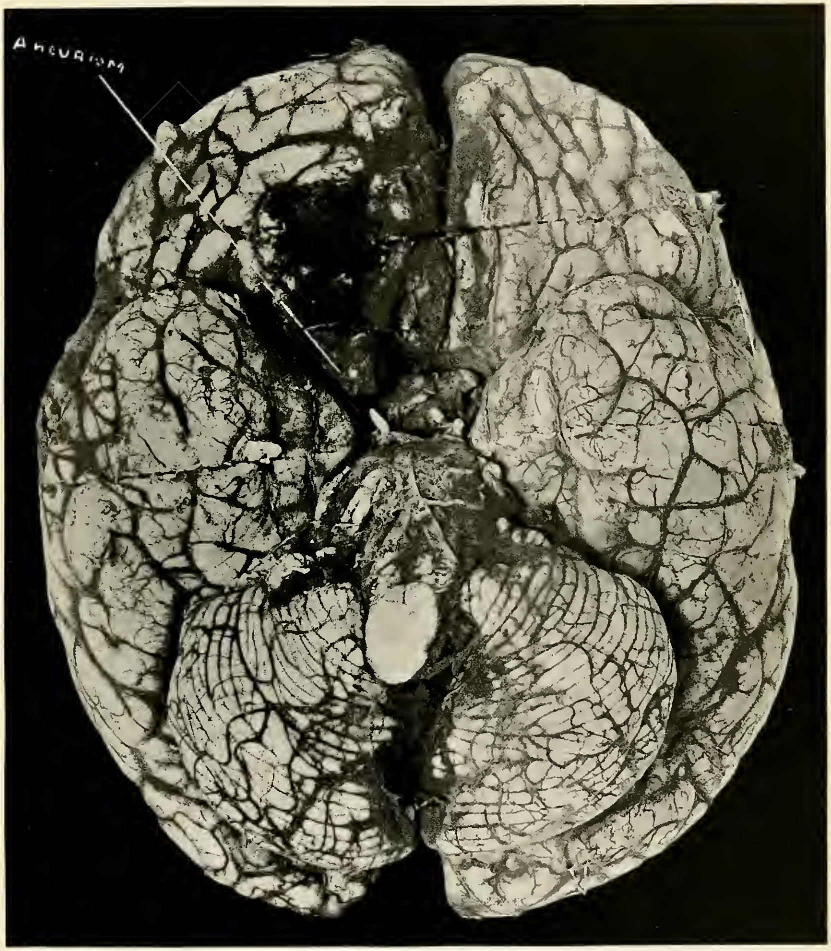

Fig. 1. Aneurism of the Right Middle Cerebral Artery

The aneurism is well shown. The dark area on the under surface of the right frontal lobe shows where the cortex had ruptured from the pressure of the subcortical clot.

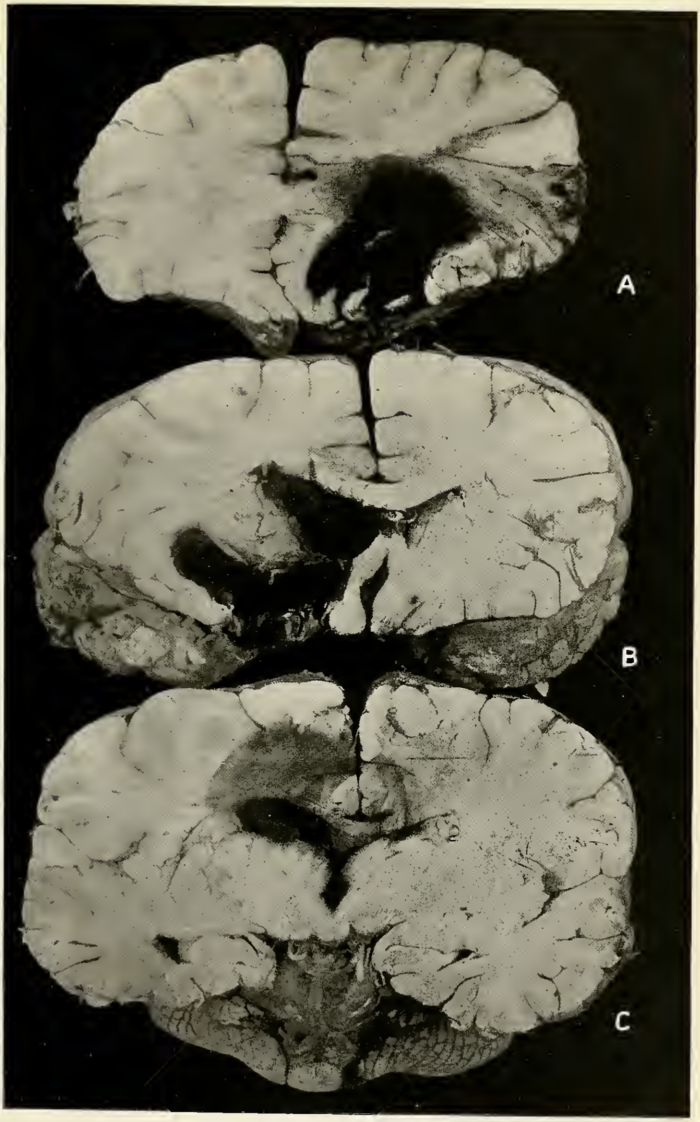

FIG. 2. CROSS SECTIONS OF THE BRAIN

Showing: *A*. The enlargement of the right frontal lobe; large area of blood in the right lobe and location of the rupture on the under surface of the right frontal lobe. *B*. A large amount of intracranial clot is still shown with the right lateral ventricle filled by blood clot. *C*. The lateral ventricle is still filled with clot and the third ventricle is seen distended by blood clot. (Note that *B* and *C* are reversed.)

There is no cyanosis or emaciation. Pulse between forty-eight and sixty-eight. Temperature and respiration normal. Blood pressure: systolic 115, diastolic 90.

Facial innervation equal. No ocular palsies. Pupils equal, moderately dilated, respond somewhat sluggishly to light. Fundi show beginning papilledema, right and left. Tongue protruded in the midline. Sense of smell normal, right and left.

Upper extremities: Power good, right equals left. Reflexes in upper limbs somewhat exaggerated, right equal left. No ataxia or incoördination.

Lower extremities: Power normal, right and left. Knee-jerks sluggish; ankle-jerks cannot be obtained. No Babinski or other pathological reflex.

Abdominal and cremasteric reflexes present and equal.

On the evening of December 29, the day of the patient's admission into the hospital, a lumbar puncture was performed, but only thirty cubic centimeters of bloody fluid were withdrawn, as the patient then complained of violent headache, faintness and dizziness. The Wassermann test of the fluid was again negative.

January 3: The condition of the patient remained unchanged until January 3 (three weeks from the beginning of the symptoms). He then began to complain of blurring of vision in his right eye, and began to have irregular fever up to 101° F. The left knee-jerk was now livelier than the right. The abdominal reflexes were diminished on the right side. The right pupil was now widely dilated and did not respond to light. The left pupil reacted to light and accommodation. There was now a papilledema of two diopters in the right eye, and a slight swelling of the left optic disc. Although it was very difficult to have the patient coöperate, it was fairly certain that vision in the nasal half of the right visual field was lost.

About midnight, the patient had a severe convulsion involving the left arm, left leg and both sides of the face. After the convulsion, there was a distinct weakness of the right lower facial muscles. For about one-half hour after the convulsion he could be aroused and during this time he talked intelligently; he then lapsed into a semicomatose condition.

January 4: The patient was in stupor and could not be aroused. The epigastric reflex was stronger on the left side. The left knee-jerk was livelier than the right; the ankle-jerks could not be obtained; there was a doubtful Babinski on the left.

The diagnosis lay between a tumor near the lateral ventricle with hemorrhage and a ruptured aneurism in the anterior or middle cranial fossa on the right side.

At 2 p. m., January 4, a bilateral subtemporal decompression was performed (Dr. Elsberg): Very little ether anesthesia was required. 1. Typical right subtemporal decompression with a large boneopening (6 x 7 cm.). Dura plum-colored. When the dura was incised, a considerable amount of old fluid blood and clots were evacuated. Exploration in all directions failed to locate the source of the hemorrhage. The right lateral ventricle was punctured, but no fluid was obtained. There was only very slight bulging of the brain tissue into the wound. 2. Left subtemporal decompression. Dura normal, cortex of brain normal, no hemorrhage on surface of cortex, no increase of pressure. Aspiration of left lateral ventricle withdrew a considerable amount of bloody cerebrospinal fluid that had the same appearance as the fluid that had been obtained by lumbar puncture. Closure of wound in the usual manner.

The patient did not improve after the operation. He remained in coma and died about eight hours after the operation.

January 5: The autopsy was performed twelve hours after death. The calvarium was normal. When the dura was incised, the right cerebral hemisphere, especially over the frontal, temporal and parietal lobes, was covered by some old fluid blood and clots. There was no blood over the convexity of the left hemisphere. The under surface of the right frontal lobe was covered by an old blood clot. In the horizontal portion of the Sylvian fissure near the median line, and impinging upon the right optic tract, was a small aneurismal mass the size of a cherry, connected with the middle cerebral artery near its origin. Careful dissection showed that this was an aneurismal sac derived from the middle cerebral artery which had ruptured into the frontal lobe and had caused a collection of blood in the lobe. The mass of blood had ruptured through the basilar surface of the lobe and had thus spread over the right hemisphere. The right lateral ventricle was distended with blood clot, as was part of the third ventricle. The left lateral ventricle contained thin bloody fluid. (*See* Figs. 1 and 2.)

SUMMARY

This case is interesting from a number of standpoints. In the first place, the symptoms all pointed to a lesion in the right hemisphere affecting the right frontal lobe and impinging upon the right optic tract or optic nerve. In this way could be explained the left pyramidal tract symptoms, the blurring of vision on the right side, the right choked disc and the defect in the right visual field. As no fluid was obtained by aspiration of the right lateral ventricle, but bloody fluid was withdrawn from the left lateral ventricle, it seemed probable that the right ventricle was either filled with clot or was occluded by pressure.

The appearance of acute cranial symptoms in a previously healthy man, with immediate rigidity of the neck, Kernig, and bloody fluid by spinal puncture, pointed very strongly to a ruptured aneurism. The importance of this syndrome of symptoms has been emphasized by many writers. In our case we were not certain whether we had to deal with a ruptured aneurism or a small neoplasm with a hemorrhage into it.

BORDERLINE PSYCHIATRY

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As recently pointed out in a previous number of the Bulletin by Drs. Hallock and Osnato very little in the way of systematic work has been attempted in borderline psychiatry and we feel that this problem has been neglected in neurological clinics. Much can be done to help these patients adjust themselves in relation to their social conditions and they can be rendered self-sufficient and self-supporting, instead of becoming derelicts floating from one clinic to another without obtaining relief.

In this group are included the various depressions, early dementia precox, some paranoid conditions and the various neuroses all representing types of social maladjustment and maladaptation.

Now we have learned that adaptation represents by its success or failure the ability to meet the various issues of life as represented by our complex, often artificial modern civilization. Even the simplest organisms in their struggle for existence have issues to meet and as the higher levels of evolution are reached life becomes more complicated and intricate and demands greater effort for successful adaptation. This failure of adaptation manifests itself by constant strivings for the unattainable and lack of contentment with things as they are. We all have our dreams of what we would like to be and what we would like to have, but most of us have to be content with fulfillments in a much less extravagant form than our desires have represented. I believe that day-dreaming, if it does not replace healthy activity, contributes not a little to our success in life, because it holds out a goal for which we are constantly reaching and striving. When repressed, these wishes are not suppressed, they are merely refused admission to consciousness in their ordinary form. They reappear in various disguises chiefly as symbols. In our ordinary life symbols are symptoms and sublimations. Thus patients often present themselves with headaches which cannot be explained on purely physical grounds, and on analysis they represent conflicts which are the real cause of the patients' illnesses. Again various fears will be found based purely on psychogenic grounds, and with a discovery of the disturbing conflicts the unwarranted fears disappear. From biological experience we learn that self-preservation and the preservation of the species are two of the greatest social instincts. As MacCurdy has recently stated in an article upon this subject, "All permanently unconscious wishes are individualistic; sublimations consist in the union of social and individualistic aims. A man may build tunnels through 'Mother Earth'; a woman may do religious work in the service of her 'Divine Father'. In every case the activity represents symbolically the indulgence of the unconscious in a way to escape censorship and, at the same time, benefits mankind." We know that no one succeeds in his work if his heart is not thoroughly in it. When we are sad we cannot work as well as when happy.

In the psychoses one sees depression when the unconscious wish strives for expression. When sublimation results elation is present. Depressions are often reactive in character and frequently represent a repression of the unconscious wishes, but this may be regarded as a protective attempt on the part of the personality against the destructive unconscious forces. This repression explains the lowering of energy and initiative in these cases, if it may be assumed that the energy is expended in the great task of repression. In the mind there are evidently destructive as well as conservative forces; the former being purely non-adapted egotistic tendencies of the unconscious, the latter the more adapted social tendencies. It seems that in manic depressive psychoses, in the benign psychoses as Hoch calls them, it is the conservative forces which have the upper hand. It has been found by Hoch in a study of the benign psychoses that the content of them was characterized by their adult level. While infantile desires of a certain type may appear in some forms of benign psychoses they are never seen in depressions, but are there repressed, while superficial ideas are substituted. In manic states they are also absent but adult sublimated derivatives are permitted to enter consciousness. It seems probable then that when depressions are produced by precipitating causes which represent fulfillment of an unconscious wish, this is always on an infantile level, but the wish-fulfillments contained in the precipitating causes of manic attacks are on an adult level.

In dementia precox there is a distinct relation to the psychoneuroses and it is often difficult to make a correct differential diagnosis. It is possible to formulate all of the mental symptoms in the same way that they are formulated in the psychoneuroses and they are capable of interpretation solely at the psychological level. It must not be lost sight of, however, that recent investigations are tending to show more and more that there are distinct biochemical disturbances during life and that pathological changes are found after death. These pathological findings must be conceived as being correlated with the psychic symptoms. The toxic theory fails to account for the form that the mental manifestations take and this is particularly so with regard to the hallucinatory trends which often appear to be external projections of unsatisfied, unconscious wishes. It would seem that the schizophrenic splitting of the personality is more profound than in the so-called functional disorders and that it goes to deeper levels, in that the infantile and the archaic in the individual have been less efficiently sublimated and so control the thoughts and acts more effectively. At present we are unable to make any specific correlation between the physical findings and the mental symptoms but it is quite possible to reconstruct the psychosis purely in psychological terms. Dementia precox is therefore to be regarded as a certain type of reaction to a mental conflict which results in a splitting of the psyche and the bringing into consciousness of unconscious mental trends. The patient is therefore confronted with an unpleasant situation to which he is unable to adjust himself adequately, and as the situation is unacceptable and impossible he is therefore driven away by his incapacity to assimilate and is cast back upon himself. The conflict of the opposing forces produces the picture which is the outward evidence of the effort on the part of the individual to reach a solution of the difficulty. The symptoms are the result of the appearance of the unconscious trends distorted and disguised.

Friedmann has demonstrated how paranoid ideas in given cases might develop as a result of unpleasant situations in the patient's life. These delusions are easily understandable when all of the circumstances attending them are uncovered and explained. Gierlich has demonstrated that persecutory ideas are often concomitants of fluctuations in affect which are manifestations of a manic depressive attack, and that many of the paranoid conditions which were associated with only slight affect disturbances, belonging to the manic depressive psychoses, might easily be overlooked and be mistaken for a real paranoid condition. In this manner a large number of the so-called acute paranoid conditions may be accounted for, but as the affect disappears the accompanying ideas vanish. Bleuler believes that paranoid conditions take their origin in certain constellations of ideas or complexes and that these are precisely of the same nature as are found in normal individuals. The psychosis develops as a result of the inability of the individual to reach an efficient adjustment to this complex. The delusion is only one expression of the disease and the mechanism involved has to deal with the content which is only the outward expression of the submerged disorder. It expresses an effort on the part of the individual to reach a satisfactory adjustment and to render the existence of the painfully affected complex more endurable. Individuals who are ambitious but who lack the ability to succeed often develop the idea that lack of success is due not to their own short-comings but to outside interferences. Thus they unconsciously create a situation which at once explains for them their difficulty and enables them to get along more satisfactorily for a time at least.

If the various conflicts which are the sources of the patient's difficulties can be unearthed and discussed freely with the patient, thus modifying his mental trends by a process of readjustment and reëducation, much can be done to benefit these so-called "borderline cases." The whole process consists chiefly in a readjustment of his mental life so that he is able to understand his conflicts at their real values. He is thus in a position to assume a healthier attitude toward life in general and particularly in regard to himself and his real purposes in life.

By coöperation with teachers and parents in the early life of the child much can be done to avoid the pitfalls of adolescence and of later adult life.

While analysis and uncovering of the painful situation is the thing most desired, it is not all that is required for the successful treatment of this class of patients. We must extend a helping hand not only by our advice, but we must aid these individuals in a practical way. If the home environment is a disagreeable one, we must strive as much as possible to make it more satisfactory. This often means the cultivation of the interest of the other members of the family, who have often come to look upon the illness of the patient as assumed for some ulterior motive, and for this reason the latter is often treated with scant courtesy. If the patient's occupation is distasteful to him and does not give him a proper outlet for his interests, an effort should be made to secure more congenial employment. Frequently these patients have talent in an artistic field which, if cultivated, solves the difficulty under which they are laboring. I do not mean to convey the impression, however, that these patients are to become dependent, but rather to emphasize methods which offer a more certain path to independence.

In trying to aid these patients the existing philanthropic organizations should be made use of as far as possible, but there will be found a number of patients who require a more intimate contact for their successful adjustment. In such cases the services of a social service worker are indispensable. Such a worker can aid in studying the environmental conditions from an individualistic standpoint. An attempt to understand the reactions of these individuals is frequently attended by gratifying and lasting results.

The following cases will serve as two types of abnormal mental reaction in individuals who might properly be said to be suffering from a borderline psychosis.

CASE I.[1] Patient: L. R.; female.

HISTORY. *Present Complaint.*—The patient states that three years ago she began to act in a peculiar way and became involved in a love affair. She believes that she was infatuated during this time and is now anxious to be treated so that there will be no recurrence of her difficulty.

Previous History.—Her employer states that she has known the patient for about a year and during this time she has worked as a housekeeper. She is a very efficient and willing worker but has always been impulsive and self-opinionated. She is usually happy but at times seems rather depressed. When the informant first knew the patient she talked about a love affair continually, wrote long letters to the object of her affection, tried in various ways to see him and made herself a general nuisance. She, however, showed nothing else that was unusual in her general demeanor and after a time she dropped the affair but continued to talk about it a great deal.

Family History.—The informant knows very little about the patient's family, but she believes they were fairly prominent in the South prior to the Civil War.

GENERAL EXAMINATION.—The patient is intensely egotistical, constantly refers to her family as being very prominent, speaks of her intellectual ability and of her virtues and details at least two senseless love affairs. She tells of having been engaged at the age of seventeen to a young lawyer. He became jealous of the attentions of other young men and, as she refused to discontinue their acceptance, the engagement was broken. She says that she was much depressed over the affair and has never seriously cared for any other man since then.

Three years ago she met an old gentleman, "seventy years of age, but well-preserved and as fine a looking man as you ever saw." She further states, "he wanted to settle money on me to marry him, but I believe in marriage only for love. After he had laid his proposition to me I began to get silly and foolish. My will power was entirely destroyed when I was in his presence. He even kissed me, but I was powerless to prevent it. I am a correct woman and I have shed oceans of tears over it, but I would let him do it again because I could not help myself. I loved this man and I was wildly infatuated with him and wrote him silly letters telling him this." She is unable to give any detailed information about his general appearance, nor does she remember his address except that he lived in a rooming house on Seventy-second Street. He told her that he was worth several hundred thousand dollars. While she feels that he really had some influence over her she does not think that he hypnotized her. After she had written a number of letters to him he dropped the affair, and with this she came to a realization that she had been acting in a manner that was entirely foreign to her and became herself again. She grieved over her humiliation for a year.

Two years later she met a middle-aged man under similar circumstances. He was a widower with two young children and, according to herself, this fact attracted her to him. She further states, however, "I took him just like a toy to amuse myself. I had no notion of marrying, but I think he wanted to marry." Again she became infatuated, wrote silly letters to this man and constantly annoyed him by calling him on the phone. Her annoyance became so great that the man finally told her that if she did not cease her attentions he would have her arrested. With the above threat she again came to the realization that her conduct had been unusual. She further stated, however, that while in her infatuated state she saw this man's face every night for several hours before falling asleep and even at present she sees his face under the same circumstances. At times she has seemed to hear his voice talking to her and even since the termination of the affair she has heard him call her name and say, "I never intend to marry." She talks in a fairly glib way yet gives the impression that she is possessed of only superficial knowledge. She constantly refers to herself as being a virtuous woman and desires to impress everyone with this fact. She is extremely garrulous so that it is difficult to interpose questions during the examination.

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[1] Presented at the Clinical Conference, Nov. 26, 1917.

She shows no psychomotor unrest. There is no sound association nor flight tendency and her trend appears to be limited to these love affairs which she relates in great detail and apparently with much satisfaction. It might be added that she had her menopause eight years ago and no change either mental or physical was noted at that time.

Physically she is an obese female of middle age, matronly in appearance, but not prepossessing nor even attractive. The physical findings are entirely negative.

Summary.—We have then a patient of middle age who is described as being of an open, lively, garrulous make-up. Three years ago she became enamored of an elderly man and constantly endeavored to force her attentions upon him. Suddenly she realized the abnormal character of her conduct and became depressed and humiliated. This affect lasted about a year. Two years later she became involved in a similar affair with a similar ending. While she is quite garrulous she shows no flight tendencies nor distractability and there is no psychomotor unrest. She expresses no paranoid ideas, but there has evidently been a definite hallucinosis of a visual and auditory nature. It is important to emphasize the fact, however, that this hallucinosis has been definitely circumscribed and there has been no evidence of any other hallucinations. Her mental organization is intact. She is alert and active mentally and shows no evidence of deterioration at the present time. It would seem that the mental condition with which we have to deal represents the fulfillment of an unsatisfied wish for marriage. There appear to have been definite mood deviations and it would seem that during the episodes of infatuation she must have been overactive and overtalkative with a diminished sense of responsibility and critical judgment.

A deviation of the manic depressive type would have to be considered because of the mood-swings and because of the egotistical, self-satisfied and garrulous make-up. Dementia precox could be ruled out because of her make-up, the age at which the condition developed, the absence of emotional deterioration and of any definite paranoid trends. She also appears to have definite insight into her condition. An early paranoic condition would have to be considered because of the fact that one feels that her difficulty is a projection externally of her unsatisfied and ungratified wishes and it is a question whether in the future she might not convert her projections into a paranoid trend, representing them as being actually fulfilled and rendering her situation in life more acceptable to her. At the present time, however, I feel that in describing this condition as a manic-like reaction we are going as far as is justified. The outcome of the case will either confirm or disprove the diagnosis. In the meantime we are trying to bring the patient to a realization of the real cause of her difficulties and she seems in a fair way to accept the explanations offered, but not without a certain amount of reluctance.

DISCUSSION

Dr. S. P. Goodhart.—While this patient's history and present mental picture do not enable us to classify her among the recognized types of the psychoses, in all probability she is a case of manic depressive insanity. Her present state is that of hypomania, now probably merging into her only comparatively normal state. Her really active periods are probably those of varying duration in which, as shown in her history, she dashes boldly and with intensity of mental and physical overaction into various affairs, the love episodes being her star performances. The insight that followed the clearing is by no means at variance with what we so frequently see in this type of psychoses to which she belongs.

Dr. Charles A. McKendree.—This case interests me very much indeed. It corresponds to some of those that we see in practice and also here in the clinic, and which we are forced to include in the undifferentiated depression class. Such cases are often considered normal, but in reality they present trends parallel to manic depressive or paranoid states. Dr. Leahy's presentation was very fine and his description of the patient's make-up perfect. Here in the Conference she manifested insight, no delusions or hallucinations, understood what she had done and the absurdity of her actions; but she showed a marked degree of perseverance and a paranoid adherence to the idea which she desired to express from which it was impossible to distract her. I believe her experiences might well be interpreted as part of either a paranoid or manic state.

Dr. Michael Osnato.—There are many prototypes of this lady throughout the country—the poor, overly-genteel women who talk as she does, have had financial reverses, always claim to have come of a splendid family and always, according to them, are possessed of superlative intellectual qualifications and of highest moral feeling. I think we have here a constitutionally inferior individual, according to the State Hospital classification; these are the cases which exhibit defects in the emotional and volitional spheres. She began at the age of seventeen to show indications of not being able to adjust herself to life. She shows tremendous emotional deterioration. Any woman approaching normal upon being asked to recount such difficulties as hers before this audience would have regarded it as an ordeal; but apparently she enjoyed the experience and was anxious for the opportunity to accuse and justify herself. This shows a judgment defect also. To my mind, she shows no characteristics of manic depressive insanity; she is not distractible, for even pointed hints from Dr. Leahy did not divert her from her discourse. She shows no motor hyperexcitability. She has none of the symptoms that correspond to manic depression. She is not even depressed, as we understand the word. There is no psychomotor retardation; it is not difficult for her to think. She has no mental pain. She shows one precox-like reaction, and that is in the idea that she was influenced by the man she met in the park. She fits into a group of cases described by Kraepelin as paraphrenia, characterized by mental deterioration and a paranoid trend. I think we would have to search away back into the early childhood of this woman to find the beginning of her trouble. One may be able to tell definitely when a typhoid or a pneumonia begins, but in cases such as this the onset, if there is such a thing, is insidious and extends back a great many years. I think Dr. Leahy will agree with me that there are probably many significant things about this patient's early life that have not been brought out.

Dr. C. J. Slocum.—I think it is difficult to differentiate between manic depressive psychosis and paranoic conditions in the dementia precox class. It seems to me that this woman presents a paranoic condition which is evinced by her claim that unseen influences are about her, the marked egotism she displays and her lack of secretiveness together with a very elaborate line of reasoning. She has apparently quite a marked idea of persecution but she shows, to my mind, no manic depression.

Dr. Adolph Stern.—The patient is far from being in a normal condition; she does not seem to me to show good insight into her exalted periods. It is a question whether she is manic. The episodes may have been manic manifestations, but she impresses me as being of the paranoid type rather than manic. Whether she is now in a manic state, which is usually one of long duration, I do not know, but her condition might as well be termed paranoid as manic. Further inquiry into her previous life might perhaps clear this up.

Dr. Sylvester R. Leahy.—Regarding paraphrenia, I understand these cases have definite trends and the end state is characterized by deterioration. This woman has definite manic symptoms and she has no deterioration whatever. It seems to me that one can get a better idea of the onset from her narration than from observing her early life as an outsider. As to whether she will have other attacks, I am not prepared to say, but her conduct today would seem to me to indicate the possibility of a recurrence of her exalted state, which may be beginning now. It seems to me there can be no doubt of the fact that this is a case of peculiar personality with manic trends.

The following case represents a repression in reaction to an unsatisfactory outcome of a love affair without any further attempt at readjustment at the present time. Undoubtedly later some sublimated derivatives will be permitted to enter consciousness and the productions of the patient will probably represent the fulfillment of her unconscious wish.

Case II.[2] Patient B. P.; female; age 20; single.

History. *Present Complaint.*—The patient was brought to the clinic by her relatives with the statement that five weeks previously she had become mute, refused to eat and would not dress herself.

Previous History.—Her early life has been uneventful. She is described as having been of a lively open make-up, and has been apparently a steady efficient worker. She has had no previous attacks. She has been employed in a factory where she met a young man for whom she developed a liking. Five weeks ago this young man left the shop, whereupon she became mute, refused to eat and would not dress herself. When told to do so she obeyed commands as to eating and dressing. She has shown no mood deviations. At all times she is apparently in contact with her surroundings and observes what goes on about her but takes no active part in it. At times she is seen looking at books and papers. So far as can be ascertained she does not hallucinate. She has been very cleanly in her habits and personal appearance.

Family History.—Entirely negative.

General Examination.—The patient assumes an easy attitude and appears to be alert for she watches everything, shifting her eyes quickly in order to observe what is going on about her. She answers no questions, not even acknowledging by a nod of her head that she understands. She obeys all commands, even intricate ones promptly and correctly. She shows no resistance to passive motion nor does she hold constrained attitudes in which she is placed. She shows no change of facial expression, but looks at the examiner with an apparent air of latent hostility. She shows no active opposition to the examination at any time. From time to time during the examination she expectorates into her handkerchief. She shows marked reaction to flash-light and pin-prick.

Physically, the patient is a well-nourished and developed young female, and all findings are entirely negative.

A week later the patient showed practically the same symptoms except that when asked what the trouble was she once said rather sharply, "I don't know," and again made the same answer when asked if she were worried.

Summary.—We have then a psychosis occurring in a young girl who is described as being of an open, lively make-up. Following the development of an attachment to a young man and concomitant with his leaving his place of employment, she develops mutism but otherwise apparently remains in contact with her surroundings except that there is an absence of initiative. Because of the acute onset of the attack with a definite precipitating factor and because of the character of the reaction presented, without evidence of fear, indifference or hallucinations, I feel that this case should be placed in the manic depressive group. Dementia precox would, of course, have to be considered, but this can be ruled out by her make-up, the acuteness of the attack and the character of the mutism which she presents. An hysterical mutism would also have to be considered, but she has never given any evidence of an hysterical make-up and the symptoms which she presents at present without any mood changes are too consistent to be purely hysterical. It is felt that the patient may subsequently develop a manic phase in which the character of her productions will show some attempt at the sublimation of her conflict.

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[2] Presented at the Clinical Conference, Jan. 28, 1918.

SOCIAL MALADJUSTMENT
A COMPULSION NEUROSIS IN A YOUNG MAN TURNING THE SERIOUS ACTIVITIES OF HIS LIFE INTO BIZARRE DISTORTIONS

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"When I see something and cannot make out what it is, I have got to go and find out—everything. For instance, before I leave the house in the morning I will look in the glass containing water to see if it is empty or if water is in it, or I will look in the coffee cup in the same way. I must make sure whether it is half-full, or quarter-full, or just how much is in it." These are the first words which the patient used in describing his complaint, and are the beginning of a long story that will be elaborated later. I first saw him in 1915. He remained under my observation for a very short time, then suddenly stopped coming to me. He reappeared in March, 1918.

These cases, as is well known, are difficult for the neurologist. There are painfully few records of such cases that have been carried through to a cure. This is much to be regretted as this particular type of case offers a specially fruitful field for study. Investigation, however, is difficult, not only because of the lack of intimate and definite knowledge of the nature of these cases, but also because the compulsive, erratic behavior of the patient makes it very difficult or prevents him entirely from keeping his appointments with the physician. No doubt, as these cases become better understood, means may be found to circumvent the difficulties standing in the way. For instance, if they could be recognized in the formative stage, before the severe symptoms of the neurosis developed, and while the means of adjustment is still plastic, more could be hoped in the way of coöperation. This, however, presupposes an intimate knowledge of the nature of the case on the part of the physician, and it is with the idea of suggesting some of these problems that the facts of this and similar cases are presented.

Case No. 22080, E. G. male, age twenty-three, single, Hebrew born in Russia, a clerk by occupation. The patient complains of certain compulsive behavior, the nature of which has been already suggested and will be subsequently described in detail.

The *previous history* is practically negative. He does not know of any previous illnesses, except an operation for appendicitis in 1913, nor is there a history of trauma. There were no convulsions in childhood, nor does he remember ever having had any infectious disease. His physical condition has always been excellent; he never complained of edema, dyspnea, tachycardia, cough, abdominal pains, disturbances of the alimentary tract or renal system. There has been no severe shock or fright, and sleep has been normal. Venereal diseases are denied. Habits are normal; he does not use drugs, alcohol nor tobacco, but drinks coffee and tea in moderation.

The patient's education began with a Rabbi at his home in Russia at the age of five. He states that he was very jealous of other boys if they did better work than he did. He made friends with other boys and had the usual playmates. He came to the United States at the age of twelve with his parents and began school here a short time after, continuing until he was sixteen. Of this period he says, "I wanted to go to school and learn but my people compelled me to peddle; I could not do my lessons as I had to peddle. I envied other boys. In order to get lessons I had to copy them from other boys and try my best to get along, but they objected to my doing this. The peddling interfered with my studying. I had no chance to work in the right way." The patient was twice left back in school.

At sixteen the patient went to work peddling for himself. He earned $3.00 a week or less peddling candy. He continued this for six months and then sought work in a shop of some kind. "The boss asked for my working papers; I fooled him that I had them, but when he told me to write something, I could not spell correctly. I tried to get working papers from the school without result. Then I worked in a clothing place pulling stitches out of cloth. I earned $3.50 a week and stayed there six months. I did not like it. Then I was with a clothing manufacturer for a while. I did not like this because I was not able to write well. Then they put me at trimming but I did not do this work right. I earned $3.50. Then in a shoe-polish manufactory as a packer for two weeks. This was dirty work; I earned $2.50. Then I was peddling for a few weeks again and after this I was idle for two months or more, then in a dry-goods store where I earned $2.50 a week. I was there for three years. I hated that. The place where I am at present is a dry-goods store; I have been there four years. At first I earned $7.00 and now $14.00 a week.

"When I saw I could not get ahead in school I wanted to get out. I was always the biggest in the class and I was ashamed. I wanted to learn a trade but my mother prevented my working on Saturday. I am sorry I let her influence me. I worry about that; I have no trade and what will the future be for me?"

The *family history* has no bearing on the case. The father died of heart disease, and the mother is living but is said not to be well, although the nature of the disease is not known. There are two brothers and five sisters all living and well. The patient has a cousin (on the father's side) who is in an institution for the insane, but beyond this point no further details as to the family history could be elicited.

The *physical examination* was negative. The voluntary motor system, nerve status, general sensory system, cranial nerves and head, and cranial morphology were normal, as was also the general systemic examination.

The Wassermann and urinalysis were not made.

There was nothing to indicate any organic complication and the diagnosis of compulsion neurosis was made.

The patient's story is as follows:

July 23, 1915: "Five weeks ago I went into a candy store to get a drink of soda water. I saw a pail of water there, and had the impulse to put my hand in the water. It was impossible for me to do it because the man would object. It was late and he was about to close the shop. I thought to myself 'It will be impossible for me to touch the water the same night' and I began to get nervous and short of breath. I went outside and stood near the store hoping to get some idea as to how I could succeed in touching the water. At last I did get an idea. I asked the storekeeper if he would give me a little water to wash my hands; I was satisfied to take it from a glass. When this satisfied me I immediately wanted something else. When I am satisfied with one thing I want something else. I wanted to touch the soda fountain, for instance, and after I did so I closed my eyes and turned away so I shouldn't look at anything else, for if I did it would draw me over to it, pull me over to touch that too. I'd have to touch anything I'd see. The man thought I was peculiar. He looked at me and I knew he was thinking something about me, but I didn't care so long as I satisfied myself. That night while I was undressing I thought of the store again. While I was in the store I glanced at something else but was not sure what it was. I was so anxious to go down and find out what it was. I tried with all my mind to overcome the foolish idea, tried to think of some serious thought and I fell asleep. On the following day I thought to myself I'd never go near that candy store again, but whenever I passed any store where they sell soda water I would go in and ask for a glass of soda. If they give me a glass that someone has just taken a drink from I cannot drink from that glass. On a certain occasion I did drink, but I didn't want to and it bothered me. The idea is that I am second. I must be first or third. On this occasion I got so nervous and excited; I thought that it might be possible for me to take two little boys along with me and give them a drink from the same glass then take it myself when I'd be the third and would be satisfied. Sometimes the same thing would bother me for three or four days.

"About a month ago I visited a male cousin. He made teasing remarks about my behavior and I got to hate him. There was a girl there and she liked him, of course, better than she did me and I did not like his making fun of me. Since I was there I began to feel bad. All these things used to refer to me only, but lately what somebody else does bothers me too. Now I worry about other people's business. When I hear someone is going to drink some soda water I follow them up to the place and have got to know what kind of soda water they are going to drink. I must stay around until I find out. The man must say it a couple of times. Worse yet, I must now know how many swallows he takes. If he takes one or three or five or seven I am satisfied, but if even numbers—two, four and so forth—I am not satisfied and I can't go away, and I must watch the next party. I stand there and stand there and can't leave. If there are several glasses on the counter I want to make sure which glass has been used first. No matter what business I have on hand I must stay. One day I drank from a glass, then I saw the shopkeeper give another soda out of the same glass. I had to stay there until still another came and got soda out of the same glass, making three. But they must be three men, not women. Certain things it must be men and certain things it makes no difference.

"I would not take a bath second—it must be first or third. I prevented my brother from bathing this morning on this account. I must rinse the bathtub twice before bathing and the third time I use it for bath water. In touching the water to get its temperature I must touch it once or three times. If I touch the tub it is the same. If some one else is taking a bath I must go in and touch the water. I must maneuvre to touch the water in the tub. To do this I'll go in just as they go out of the bathroom, or manage it somehow or other. If there is water in the bottle in the ice box and some is missing from the bottle I want to know who drank from the bottle first. At table I must have my own glass—I keep it in a special place. There are two glasses just alike—once they were mixed and I got terribly excited. I would have given anything I possessed to know which was which. My mother and sister had to convince me which was my glass. Since then I broke a piece out of the glass to make sure.

"When I am in the street, if I see someone carrying something, I must find out what it is. I have the impulse to go to them and ask what it is they carry. I have done this many times. When they are carrying a vessel such as milk or water or beer is carried in, I am worried until I know what they have. When I think I can guess it, for instance, if I know the people and am pretty sure what they have, I am not upset, but when I don't know them I am compelled to go and ask them. If they don't give me a straight answer I have to ask again. If I say, for instance, 'What have you there—beer?' they may answer 'What does it look like?'. I must then ask again, 'Is it beer?' and if they say 'Sure it is beer,' I am satisfied.

"When I see certain things in the street I have got to pick them up. That is, if I cannot make sure what they are. If in some way I can know just what they are and know I can get them any time, it does not trouble me, but if I am not sure I have to make sure. And if I start picking up one thing I have to pick up things all about me. (The patient pulled some odds and ends of papers out of his pocket which he had to pick up. His room, he says, is full of "truck" of this kind.) When I have got the thing I don't want it and I don't care anything about it. I saw a piece of paper in a garage and I had to go in and get it. I felt very bad to have to do this and tried to keep myself from looking around any more, for if I should spy something else I'd have to go and get that too. Today coming here on the subway I saw from the train a bit of newspaper on the platform just as the train drew out. I went with the train a few stations and then I had to get out and go back to the station where I saw the paper and find out exactly what the paper was. When I got it I was satisfied for the time."

July 24, 1915: "I saw opposite me in the car a woman carrying a valise. I felt I must know what was in the valise. It was impossible for me to go and ask the woman in the car, she would give me hell, so I had to walk away and go into another car. Last week on a street car I looked at a man's hat. I must know what kind of a hat it is and where he bought it. To quiet my mind I walked away making believe it was a Young hat. I got so excited, my breath came quickly, you have no idea! I see a watch chain and I must ask the man what time it is—I don't care what time it is, but I must see the watch and know what kind of a watch it is.

"When this trouble first started (I was eleven years old) I used to have to go out in the street and look at the windows of houses to see whether the gas was turned out or not. I must know. When a wagon came by I started to run after the wagon until I could make sure of the color of the horses. Only certain wagons; some wagons did not trouble me. I don't know why. In a field I would see a stick; I would drag it out from the mud and bring it home; after that I did not care what became of it. When I got it home I was satisfied. I saw some hay on a wagon and had to go and get some of it. Once I went to a spring where the people used to go for water—this was when I was nine years old in Russia—I went there with a little girl who was going for a pail of water. I looked into the water and put my hand in, and I saw the bottom. This tempted me to reach the bottom with my hand and I did it. I think I did touch bottom because I was satisfied—if I had not touched the bottom I do not know what I would do. Even a couple of weeks ago it troubled me whether I touched the bottom of that spring or not. Lots of the things of this sort that have happened in the past trouble me, and I feel that I would like to go back and make sure. I sometimes wonder how the bottom of the ocean looks and get scared thinking how I can find out."

The following is an account of the patient's disturbances which he wrote out at my request:

"I must drink one of three kinds of soda water a day from one of three different fountains. Three weeks ago coming home one evening late from work, while sitting in the train I remembered that I drank only two kinds of soda water and I wondered where I could get a drink of another kind and this got me excited, because around my way there are no stands where I can get a drink. When I got off the train I looked around to see if there was a stand where I could buy a drink, but there was no stand for several blocks and as it was late in the evening I did not feel like going that distance. There was a candy store, however, one block away, but I did not feel like going to that place because I was afraid that I would become nervous and lose control of myself, as in most of the candy stores there are pails of water standing and I was afraid that I would want to touch the water in that pail. After thinking a little I made up my mind to go in the candy store but not to look around at all, but upon entering the store I saw the pail of water, and then I was very sorry that I had entered the place because I was tempted to touch the water. As it was the first time I had been in that store I did not have the nerve to ask the man for some water from the pail. After standing there quite a while I bought some things in order to wait for a customer who was in there to leave. When the customer had gone I asked the man to give me a little water from that pail to wash my hands, but he misunderstood me, and gave me some from the faucet instead of the pail. I tried to believe that I was satisfied and that it did not make any difference to me what water he gave me because the water in the pail came from that faucet anyway. I went home not quite satisfied with myself, but I made every effort to forget about it. While I was thinking about it I fell asleep. The next morning I recalled that there was only a little water in the pail of the night before and I was anxious to see if they filled the pail up with more water so I went in the store again that morning to see if they had really filled it, but found that they had not. While I was in the store I was attracted by several things and I tried to keep my attention off of them, but when I left the store and got on the train to go to work, on the way all the things which I had seen in the store bothered my mind and I started thinking of them. For instance, I saw a glass in the pail and I could not make up my mind whether it was thick or thin glass so I got very much excited and nervous and I had the impulse to get off the train and go back and see and make sure what kind of glass it was. I satisfied myself for the time being by thinking that I would find out when I got home, then I began thinking of something else that I had seen in the candy store—it was two little glasses that had just a little soda water left in them. One seemed to be chocolate, and I could not make out what the other kind was. After thinking awhile I made up my mind that the color of it was like cherry soda water, but this did not satisfy me because I know that many other syrups have the same color. All the same I decided that it was cherry. All the time I was thinking this out I was terribly excited and had to get up from my seat in the train and walk up and down the car—and could not rest anywhere, and at the same time I wanted to get out of the train and go back to the candy store and ask what kind of soda was in the two little glasses. I had a little hope that later I could get the storekeeper to agree with me that it was cherry. I thought that he would tell me when I came back from work, so in that way I was not forced to get out of the train and go back immediately.

"That day I did not have patience to work, I was very nervous thinking all day long about that soda water. I waited only for night to come so that I could get home and go to the store and ask about the soda water in the little glasses that I had seen in the morning. After worrying all day and being very anxious I became very sick, and since that day I am very much troubled if I cannot get or see a thing that I want. That night I went in the candy store to ask the storekeeper about the two little glasses of soda water, but I could not get a chance to ask him about it so I thought I would leave it until the next day. While I was in there I asked him to tell me which flavor they sell the most of. Then I asked him to give me a glass of cherry soda, as he told me they sell more of cherry soda than any other. Finding this out, I was quite sure the soda in the glass that morning was cherry, and I thought I would have no more doubt that it was cherry if the storekeeper would also agree with me. The next day I went in the store and met the storekeeper alone telling him a little of my trouble, and I asked him to agree with me that the soda that was left in the little glass on that morning was cherry soda. At last he agreed with me that it was cherry soda and I was perfectly satisfied."

I heard nothing from the patient until March, 1918. He has been drafted in the army. Although he denies that he has come to me now in order to clear himself from the draft, I do not feel at all sure that this is not, consciously or unconsciously, the real reason for his reappearance just now. The history continues:

March 26, 1918: "If I see a newspaper I have got to find out just which paper it is, the *Journal*, the *World*, etc. If it is only a bit of newspaper and I do not know what paper it is I will put it in a safe place and hope that some day some bit of information will lead me to discover what paper it is. Still if it is put away safely and I know it is safe I will forget about it. I will pick up a business card and put it in my pocket. As long as I have it with me I don't think of it, but when I can't get it or it is in a place where it is impossible for me to get it I have a horrible fear. I get restless and my heart gets weak; I have a choking feeling and I can't think of anything else but that particular thing. A terrible feeling is in my heart and I can't catch my breath. I loosen my collar; I can't find a place for myself. If I don't get it I get ideas and make believe I may have the opportunity to see that same thing once again, then it comes to me it will be impossible to see that same thing again and I get wild. If I am in the house I have to run out, I can't stay in a closed place and have to catch my breath. Once when buying a paper at a newsstand I had the impulse to see how many papers of each kind there were there. The man looked at me as though I were crazy. 'What are you doing? You're spoiling my business.' Then I started to talk something to distract his attention so that I could in the meantime count the papers, but he pushed me away. In the train if some one is carrying a package or valise I have the impulse to ask them what they have in the package. I begin wondering how I can bring this about; I'll say to the person, 'It's a heavy package, what is in it?' But if I can't do that, I get weak and have to go into a different car or leave the train.

"I have to see the trains; where this train is going, where that train is going; I turn my head up and down, this way and that and feel terrible after I get out. I get a notion I must see how the motorman looks, and have to run all the way up to the front of the train. If it is a closed box and the shades are down, I can't take a peep at him and I get restless, and try my utmost to attract his attention. If I hear conversation and do not get it all, I want to find out the rest or learn what they are talking about or what sort of words would fit in that conversation. If I know the people I will go and ask them what they were talking about and ask them to repeat what they said —I must know. If my boss sells something in the store, I must know what he sold and how much he got for it. I try to ask him in such a way that he won't suspect my trouble. I try to get a peep while the customer is in the store to see what was in the package she bought. I run about the store and feel wild.

"But water is the main thing—that gets me crazy. I am worse in summer than I am in the winter. I get wild in the summer. They drink more soda in summer. I used to have thoughts of touching things, but that is not so much in my mind now. Now it is things I want *to know*. For instance, if I know a person is drinking different flavors of soda water each day, I make every excuse to find out what it is he drinks. I get all mixed up, if he is taking different flavors, but if he is taking the same drink I don't mind. (The patient is speaking of associates in business or elsewhere.) It wouldn't bother me if I did not hear them talking about the different sodas, etc. I do my best to try not to listen. I will go to the standkeeper and ask the man to give me the same soda the last man just drank, in order to find out. He will reply, 'How do you expect me to remember?'. Then I will get very angry and nervous. Meantime others are coming and buying and I have to make up all sorts of things."

April 1, 1918: "I am ashamed to tell my employer that I want to get off in order to go and see the doctor. I try in every way to get off without his knowing why. If I am impelled to go to someone with one of my questions and he replies in a nasty way, 'What's the difference, why do you want to know?', if he suspects me, I am ashamed, and hope I don't have to face that person again. I keep turning and twisting my head in all directions until I get all tired out. Sometimes I keep looking up and go along that way, so that nothing will attract my attention."

An account of his present symptoms written by the patient is as follows:

"Almost every morning when I go to work, or go away for the day, I must look at certain things that I know or think will be gone when I get back, to see where and in what position these things are. Frequently after I have left the house all the things that I have been looking at come to my mind and trouble me and I go over and over them in my mind. I try my very best to avoid thinking about them but I can't help it. While I am on my way these things continue to trouble me. I think of them in the places where I saw them and think of the exact position in which I saw them, and if I don't remember just where and in what position I saw the things and I know that when I get back I will not find them in the same position as when I left them, I begin to feel terrible and I must turn back to see and make sure where and in what place the things are. Some mornings it gets me mixed up so badly that I feel terrible all day. On my way to work while sitting in the train I try my best to put my mind on something else in order not to think of the things at home. Sometimes I succeed in keeping my mind busy with something else, but when I don't I feel terrible. One day last week before I left the house I saw a paper in our garbage can. I had the impulse to look at it to see what paper it was, but instead of looking at it I tried to make believe that it was a *Journal* because in our home we take the *Journal*. But on my way it came to my mind that I am not sure whether it was a *Journal* or another paper, as we read another paper sometimes. I tried my best not to turn back but to make believe that the paper was a *Journal*, yet it was impossible for me to resist and I had to go back and make sure what paper it was. If that paper had been lying in another place not in the garbage can I would have thought 'I can find out what kind of paper it is when I get home' but I knew the garbage can would be emptied before I returned, and I would never know what paper it was and I had to go back.

"Sometimes while sitting in the subway train it comes to my mind that I must know how many people are going out and coming in the train, especially in the car I am in. One night this week when I was riding home, at a certain station it came to my mind that I must know how many people are going and coming in the car that I was in, so I began to count. I counted the people that came in and forgot to count how many people went out, so I had to get up in a hurry and go outside and look over the platform to try and make out the number of people that left the car, but it was impossible for me because there were so many people. I was standing on the platform thinking and figuring how many people could have gone out of the car but my mind couldn't agree. So I had to take it the best way I could, because the train had gone. Finally, I quieted my mind by thinking that the next night I would pass the same station at the same time and watch how many are going out so I can have a little idea. But, as I continued my journey, to make sure not to forget how many people were leaving and coming in the car at each station I took a pencil and paper and marked it down.

"One day last week while going to work I saw a horse lying on the street and people were trying to help it up. Suddenly it came to my mind that I must know how and when they are going to get that horse up. Standing there a while I saw that it was hard for them to get the horse up and I wished they would get it up quick, as I was in a hurry to go to work, and I was in great anxiety. While standing there some people that I know came up and greeted me, but I did not pay any attention to their greetings as I had that horse on my mind. Some of them asked me what I was standing there for, and I gave them all kinds of answers. Sometimes, as soon as I saw them, I made out that I was brushing my clothes or looking for something in my pocket. I could not go away because I was afraid that if I did go away, these things would come upon my mind as they usually do, and I would have to come back and make sure. So in this case I did not want to take any chances, knowing what the result would be, therefore, I stood there until they got the horse up. By that time it was too late for me to go to work, so I stood there until they got everything fixed up, but after I was satisfied with this, something else came to my mind and the longer I stood there the more there was for me to find out, and even then I was not fully satisfied because I did not find out everything.

"Yesterday, while I was standing outside the door, the butcher's boy came across the street to change $5.00. While he was changing the money I did not think of anything, but after he left I looked after him and saw a customer in his store and began wondering how much money she spent there. When that boy came again to the store where I work, I tried to get a chance to talk with him in order to find out how much money the customer spent in his store. Standing near him, impatiently waiting for a chance to talk with him, I asked him in a way that he shouldn't think that I was too anxious to find out, what kind of a sale he made. I said to him, 'I'll bet it wasn't worth while to take the trouble to change that $5.00 for that small sale'. I thought that he would say that it was and that he would also tell me how much money he took in, but instead he only told me that it was worth while. Then I said to him again, 'Really, how much money did you take in?' This time he kind of suspected me, or I thought he did, but he said, 'I made the sale for $2.00, and a little more.' But I still did not know the exact amount and I was ashamed to ask him any further question, so I remained unsatisfied and very restless the whole night.

"One day while I was standing outside of a store I saw a man coming out of another store nearby carrying a package. I felt I must find out what he bought so I went in the store from which he came and found out in a roundabout way what the man had bought. I was told it was muslin. But after I knew what kind of goods he bought, I was then anxious to know how many yards. Then after standing a while and thinking about this, it came to my mind that I must find out how much money the sale amounted to, and I couldn't think what kind of an excuse to give him and was also afraid that he would suspect me. When the thought came to me that it would be impossible to find out how much money the sale amounted to, I felt terrible and simply went wild. Even at night when I went home, thinking of it in the car, I had to get up and walk around—I could not catch my breath. Especially when I thought I shall see him the next day (as he is a business neighbor) my mind was very restless and uneasy. I simply hate to see him, and when I do see him I try my best not to look at him."

The impression this patient creates by his attitude and manner is worth noting. He is physically robust, stout in build, has a ruddy color, but he carries himself like a prisoner under sentence, his head drops and his eyes avoid the examiner. Sitting, he bends forward, looks at the floor or down at the desk most of the time, or glances to one side or the other. He is never in repose. There is a peculiar squint in his eye. He is mentally alert, there is nothing negative or dull about him. There is an aggressive quality in his make-up. He is quite evidently struggling against emotions of shame, of being held in suspicion, and so forth, expressing such emotions himself in such words as "I feel ashamed," "I am afraid he will suspect me," "I am ashamed to tell the boss I have to go to the doctor, and try in every way to get off without his knowing why," and many other phrases.

As to coöperation in this case I do not feel sure; if I can lead him to the least bit of insight into the nature of his condition I shall probably get active coöperation. He has intelligence and energy.

Comparing this case with the patient L. E. reported in the March BULLETIN, several striking characteristics will be seen common to both. In the present case, as in the former, the touching impulse is at once apparent. The looking impulse is equally strong in both, and the relation of the impulsions to number is also common to both and striking. A certain omnipotent character to some of the compulsive ideas, so marked a feature in the former case, does not appear to play so prominent a part in the present one, although I feel sure that it exists. While the looking and touching impulses are prominent, the impulse *to know* is the dominant one in this patient, at least at present—*he must find out.*

The "text" of the compulsion, which is so definite in the former case does not appear to be so in the present one, but I suspect that a definite text will appear here also when I have been able to study the case a little further. For example, he "must be satisfied" either in looking or touching or knowing, quite possibly this is part of his text. It is only things which are prohibited, which create the compulsive impulse in him. I asked him why he did not feel some craving to know what was hidden in a closed box that was on the desk; he replied, "I know you would tell me everything so I don't care to know; it's only where it would be kept from me that I feel the impulse." The impulse is here pitted against a prohibition; this came out strongly in the former case in a different way. The "text" may run, "If something is hidden from me I am compelled to find out what it is, then I will be satisfied," or "If something is hidden from me, I get very much excited till I can find out about it", or "If I am prohibited from knowing something, I get excited and am compelled to find out."

There is the same indefiniteness as to the time and incident of the onset of the neurosis as was shown in the first case. He tells several different experiences in the early years of his childhood as the real onset of the neurosis just as L. E. did. The fact is he cannot place it.

The nervous anxiety and tension of this patient as observed in my sessions with him were even greater I think than that of the former.

CONCLUSIONS

This patient presents a compulsive disturbance of long standing and of so severe a character that it has made a joke of his education in school and has rendered him absolutely unfit for any of the normal activities of his life. Employed as a clerk he is constantly harrowed by the impulsive ideas that he is obliged to carry out, so that he is irregular and inefficient in his work and is able to hold the position he is now in only through the tolerance of a friendly employer. All the activities of his life, serious and social—if there can be said to be any lighter side to such an existence—are turned into bizarre distortions. Drafted in the army he is totally unfit for duty, and will without doubt be released when he appears before the medical examiners.

Otherwise normal mentally, the neurosis renders him a practically useless citizen. This case is another example of the many which we ought to recognize and cure in the early years of their development.

STUDIES ON THE MORPHOLOGY OF THE GRAY MATTER IN THE BRAIN STEM OF VERTEBRATES BY THE RECONSTRUCTION METHOD—*Continued*

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PRIMATES

THE VESTIBULAR NUCLEI IN THE GORILLA

(STUDY NO. 2)

In the preceding report of this series of studies the morphology and principal connections of the gracile and cuneate nuclei were described. The comparatively simple relations of these structures, however, stand in marked contrast to those of the vestibular nuclei, which form part of a system whose connections are complex and intricate.

Clinically, it is not an exaggeration to say that the symptom-complexes produced by interference with the vestibular mechanism represent a prominent diagnostic problem of the day. Labyrinthine disease, tumors of the nervus acusticus, tumors in the cerebello-pontile angle, diseases of the cerebellum, all produce vertigo, oculomotor changes and muscular incoördination.

Broadly speaking the coördinate acts of every-day life may be considered as the result of the domination of cerebral motion formulae over vestibular or cerebellar formulae, whereas, *incoördination* is a pathological condition in which the ratio in favor of cerebral influence has declined. The vestibular apparatus is a mechanical instrument; it is primitive in its nature and but little altered by phylogenetic changes. Wilson and Pike (1) conclude that " . . . in the vestibular mechanism and its central connections we have a phylogenetically primitive nervous mechanism which has persisted throughout the vertebrate phylum." They believe that this mechanism in the higher vertebrates has come into direct relationship with newer pathways, bringing it especially under the influence of the cerebral cortex.

Herrick (2) emphasizes the fact that there is no important pathway from the vestibular apparatus to the thalamus or cerebral cortex, and that acts resulting from stimulation of the semi-circular canals are unconsciously performed. The cerebellum, however, has connections with the cerebrum—the "newer pathways" of Wilson and Pike. Cerebral stimuli are relayed to the cerebellum from the terminations of the frontopontile and the occipitotemporopontile tracts. Cerebellar impulses pass by way of the superior cerebellar peduncle to the red nuclei, and thence to the thalami and cerebral cortex (3).

If we agree with the conclusions of Herrick and of Wilson and Pike that the vestibular nuclei have no direct connection with the pallium, then disturbances of the vestibular mechanism, in order to become conscious and produce subjective symptoms, require an intermediate agency. The first of these agencies is the cerebellum. The original labyrinthine stimulus, however, in passing through this circuitous route (cerebello-rubro-thalamo-corti cal) is greatly modified by the time it reaches consciousness. If we turn back to the sense of *position* for a moment, in order to illustrate exactly what is meant by "modification," it will be realized that consciousness of position does not mean the recognition of the individual muscles, joints and tendons, *per se*, concerned in maintaining a limb or other part of the body in a certain attitude, but is the interpretation of a composite of the stimuli arising from these muscles, tendons, and joints, that eventually reaches the cortex. Thus while the posterior columns and median fillet carry muscle, joint and tendon impulses, there is a difference in the way that these impulses are combined in the two tracts. A single axone of the posterior column may carry an impulse from a particular muscle, a particular joint, or a particular tendon, whereas, a fiber in the median fillet, on account of the redistribution of stimuli in the gracile and cuneate nuclei will convey a combination of these sensory components. Professor Strong (personal communication) has calculated that ten fillet fibers can transmit impulses received from a hundred posterior column axones. The summation of normal stimuli from either the muscle-joint-tendon sense end-organs or the labyrinth is not recognized consciously as such. However, let some disturbing element interfere with a muscle-joint-tendon sense impulse, and immediately this abnormal impulse is interpreted in consciousness. In like manner the normal unconscious vestibular impulses also may arise into consciousness only as the result of some disturbing element, although such "modified" impressions as do become conscious are not referred to the semicircular canals but to the relations of the body to space, and are described as dizziness, giddiness, vertigo, etc.

When one considers the fact that a pathological impulse from the labyrinth to consciousness passes through many nuclei whose axones are connected to the motor system, it can be readily understood how easy it would be for such an impulse to become diverted and not reach consciousness at all. In Cushing's series of thirty "Verified Acoustic Tumors," (4) all except one showed signs of incoördination, and the one exceptional case complained of twitching of the left arm and leg. Mention of dizziness and vertigo was made in only twenty-three instances. Although the necessarily abbreviated form in which these illuminating cases had to be presented makes one hesitate about coming to any definite conclusion, it may be said tentatively that in this particular series injury to the vestibular nerve produced motor incoördination more frequently than it did subjective symptoms usually associated with the vestibular apparatus.

The chief complaint of a patient suffering from a disorder of the proprioceptive system is interference with *motion*. A tabetic does not say that his muscle sense is lost, but that he has difficulty in *walking;* in like manner a tumor of the acoustic nerve does not produce symptoms referable to the vestibule, but to the *gait*. Consequently the diagnosis of any case that presents incoördination as the chief symptoms requires the systematic search of the entire proprioceptive system for the source of the trouble. As the "spread" of the lesion frequently involves some neighboring structure whose unmistakable signs give a clue as to the location of the injury, the diagnostician should bear in mind the relations of the tracts he has under consideration.

In the following table the components of the proprioceptive system are enumerated in such a manner as to aid in the establishment of an anatomical diagnosis.

SUMMARY OF PROPRIOCEPTIVE SYSTEM

Afferent Pathways

A. MUSCLE-JOINT-TENDON SENSE DIVISION

1. To Pallium (3 neurones)

Neurone 1

(*a*) End-organ to dorsal root ganglion via peripheral process of unipolar cell.

(*b*) To nucleus gracilis or cuneatus, as ascending arms of central processes of dorsal root ganglion cells, via columns of Goll and Burdach.

Neurone 2
From nucleus gracilis or cuneatus to thalamus via median fillet.

Neurone 3
From thalamus to postcentral convolution via thalamocortical radiation.

2. To Cerebellum (2 neurones)

Neurone 1
- (*a*) End-organ to dorsal root ganglion via peripheral process of unipolar cell.
- (*b*) To Clarke's column or dorsal horn of cord via central process of dorsal root ganglion cell.

Neurone 2
- (*a*) From Clarke's column to vermis of cerebellum via dorsal spinocerebellar tract.
- (*b*) From dorsal horn of cord to vermis of cerebellum via ventral spinocerebellar tract.

B. VESTIBULAR DIVISION

From Semicircular Canal to Vestibular Nuclei.
- (*a*) From specialized epithelium in ampullae of semicircular canal to Scarpa's ganglion by peripheral processes of bipolar cells.
- (*b*) Central processes pass to medulla, and bifurcate into ascending and descending arms.

Ascending arms terminate in:
- (*a*) Nucleus motorius (Deiters).
- (*b*) Nucleus angularis (von Bechterew).
- (*c*) Nucleus fastigius and cortex of cerebellum.

Descending arms terminate in:
- (*a*) Nucleus triangularis.
- (*b*) Nucleus of the descending root.

The "end-stations" of proprioceptive impulses are, therefore, (1) the pallium, (2) the cerebellum, (3) the vestibular nuclei. The coördination pathways between these structures are as follows:

Coördinative Pathways

A. BETWEEN CEREBELLUM AND PALLIUM

1. Cerebellum to Pallium (3 neurones)

Neurone 1
Dentate nucleus to opposite red nucleus via superior cerebellar peduncle.

Neurone 2
Red nucleus to thalamus.

Neurone 3
Thalamus to cortex of cerebrum.

2. Pallium to Cerebellum (2 neurones)

Neurone 1
- (*a*) Frontal lobe to pontile nuclei via frontopontile tract.
- (*b*) Temporal lobe to pontile nuclei via temporopontile tract.
- (*c*) Occipital lobe to pontile nuclei via occipitopontile tract.

Neurone 2
- (*a*) Pontile nuclei to cerebellum via middle peduncle of cerebellum.

B. BETWEEN CEREBELLUM AND VESTIBULAR NUCLEI

1. Semicircular Canals to Cerebellum

Central processes of bipolar cells in Scarpa's ganglion to nucleus fastigius and cerebellar cortex.

2. Cerebellum to Vestibular Nuclei
- (*a*) Nucleus fastigius to Deiters' nucleus.
- (*b*) Nucleus fastigius to von Bechterew's nucleus.

Efferent Pathways

1. To Spinal Appendicular and Trunk Musculature (2 neurones)

Neurone 1
- (*a*) Red nucleus to opposite ventral horn of spinal cord via rubrospinal tract.
- (*b*) Deiters' nucleus to ventral horns of spinal cord via Deitero-spinal tract.

Neurone 2
Spinal motor nerves to end-organs in muscles.

2. To Cranial Musculature (2 neurones)

Neurone 1
- (*a*) Deiters' and von Bechterew's nuclei to nuclei of oculomotor, trochlear and abducent nerves via posterior longitudinal fasiculus.
- (*b*) Nucleus triangularis and nucleus of descending root to nuclei of the reticular formation and nuclei of oculo-motor nerves.

Neurone 2
- (*a*) Oculomotor nerve to end-organs in m. levator palpebrarum, rectus superioris, rectus internus, rectus inferioris, obliquus inferioris.
- (*b*) Trochlear nerve to m. obliquus superioris.
- (*c*) Abducent nerve to m. rectus externus.

Terminal neurones from reticular formation are not known, but possibly pass to neck or tongue muscles.

This summary has been graphically illustrated in Fig. 10.

A study of this table and the figure clearly shows that, no matter how complex the associations of proprioceptive impulses may be, they ultimately impinge upon the lower motor neurones, which serve as the final carriers of efferent stimuli of the entire central nervous system. An analysis of the integrative muscular action resulting from these stimuli, both in health and disease, has been shown graphically by Dr. Frederick Tilney and Dr. A. V. S. Lambert. These observers have graphically demonstrated the difference between disturbances of muscular action due to interference with the coördinating system and volitionary system. Cerebellar ataxia would be a representative of the first type of disturbance, and paralysis agitans of the second; or, anatomically speaking, the first type has to do with the vestibular apparatus and its modifications and the second with the precentral cortex and corpora striata. It is from this point of view that I wish to restate some of the well-known facts about the vestibular mechanism, and by means of the reconstruction method call attention to the morphology and relations of the vestibular nuclei themselves.

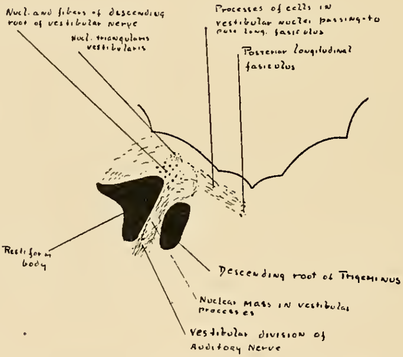
Nucl. and fibers of descending root of vestibular nerve
Nucl. triangularis vestibularis
Processes of cells in vestibular nuclei passing to post. long. fasiculus
Posterior longitudinal fasiculus
Restiform body
Descending root of Trigeminus
Nuclear mass in vestibular processes
Vestibular division of Auditory Nerve

FIG. 1. (SEC. 540.) ENTRANCE OF VESTIBULAR NERVE

The vestibular portion of the auditory nerve is composed of central processes of the bipolar cells of Scarpa's ganglion. The peripheral processes terminate in the ampullae of the semicircular canals. As soon as the auditory nerve enters the brain stem it is split by the restiform body into its original functional components, the cochlear division passing to the outer side and the vestibular division to the inner side of this structure.

Fig. 1 shows the entrance of the vestibular nerve. It passes through a strait, bounded laterally by the restiform body and mesially by the descending root of the trigeminus. A small island of gray matter is shown in the midst of the vestibular fibers, near the peripheral end of this strait. At the central end of this passageway a triangular-shaped structure, composed of gray matter and fibers cut at all angles, fits into it like a plug. One of the apices of this structure extends into the vestibular strait and receives many of the fibers of the vestibular nerve. Other vestibular fibers pass mesial to this nucleus and arch around it to reach its dorsal aspect, where they terminate in another nucleus which is in close relationship with the gray substance of the ventricular floor. Extending from these nuclei to the dorsal portion of the median raphé, another system of fibers is observed that enters the posterior longitudinal fasiculus and turns cephalad to reach the nuclei of the ocular muscles.

This section represents the fundamental pattern of the vestibular system, except that in a transverse section the bifurcation of the nerve fibers into ascending and descending arms cannot be shown. The vestibular fibers are the primary neurones. The nuclear masses receive the termination of these neurones and contain (1) cell-bodies of the second series of neurones, (2) ascending and descending bifurcations of vestibular nerve, (3) axones of the cell-bodies mentioned above, some of which pass transversely and some in a caudal or cephalic direction.

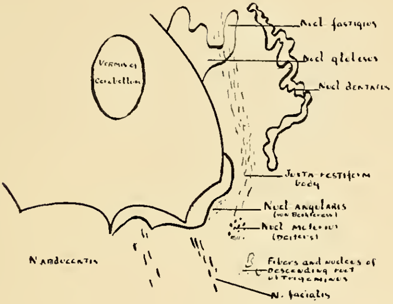

FIG. 2. (SEC. 575.) SHOWING NUCLEI OF ASCENDING ARMS OF VESTIBULAR NERVE

Sections made at a distance from Fig. 1 show the same general arrangement except that, naturally, the entrance of the vestibular nerve will not be seen. Obviously, vestibular nuclei observed in sections below Fig. 1 receive the descending arms of the vestibular nerve, whereas those above it receive the ascending arms.

Fig. 2 (1.75 mm. cephalad of Fig. 1) shows the two nuclear masses, and processes connecting them with the nucleus fastigius and more distant parts of the cerebellum.

Fig. 3 (3 mm. caudal of Fig. 1) again shows the two nuclear structures and a second neurone, arching through the restiform body to terminate in the reticular formation.

Regarding the nuclei themselves, it can be stated that they exist in pairs and extend almost the entire length of the fourth ventricle. One nucleus is close to or combined with the gray matter of the ventricular floor, the other is in the white matter of the brainstem, close to the dorsal boundary of the restiform body.

The entrance of the vestibular nerve differentiates the vestibular nuclei into a superior and inferior group; below this location the dorsal nucleus is called the nucleus triangularis, and the ventral nucleus, the nucleus of the descending root. Above this region, the dorsal nucleus becomes the nucleus angularis (or von Bechterew's nucleus) and the ventral nucleus, the nucleus motorius (or Deiters' nucleus). Deiters' nucleus is characterized by its large motor cells (also called nucleus magnocellularis).

It is of interest to note that the nucleus of the descending root appears at the cephalic end of the nucleus cuneatus, and that thus the anatomical representation of the muscle-sense type of proprioceptive sensation blends with the vestibular form.

The triangular nucleus, if continued downward, would occupy the region of the dorsal nucleus of the vagus. Following it upwards, its lateral expansion is observed to be coincident with the diminution in size of the vagal nuclei.

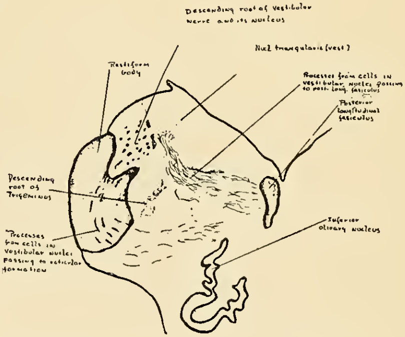
Descending root of vestibular nerve and its nucleus
Nucl triangularis (vest)
Restiform body
Processes from cells in vestibular nuclei passing to post. long. fasciculus
Posterior longitudinal fasciculus
Descending root of Trigeminus
Inferior olivary nucleus
Processes from cells in vestibular nuclei passing to reticular formation

FIG. 3. (SEC. 480.) SHOWING NUCLEI OF DESCENDING ARMS OF VESTIBULAR NERVE

At the upper limit of the hypoglossal nuclei are the nuclei intercalati of Staderini and the nuclei funiculi teres. Bárány (5) states that the intercalated nuclei have a vestibular function, and Jacobsohn (6) calls attention to the resemblance of the cells of the triangular nucleus to those of the nucleus funiculus teres.

The location of the triangular nucleus would strongly suggest a splanchnic function, and its size, as compared with the other vestibular nuclei, would further suggest that the sympathetic functions were the most important of any. The fact that the vestibular nuclei send processes to the vagal system, and that one result of vestibular stimulation is vomiting (*i. e.*, sea-sickness), is a familiar demonstration of their splanchnic connections.

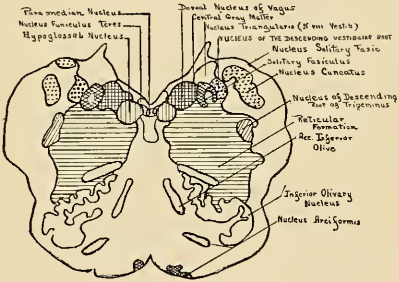

FIG. 4. (SEC. 420)

The relations that both the triangular nucleus and nucleus of the descending root bear to the dorsal nucleus of the vagus and nucleus of the solitary fasiculus suggest an explanation of the dysphagia, dysarthria and loss of taste that occurred in some of Cushing's acoustic tumor cases, yet in the present state of knowledge no evidence that shows a pathway between the vestibular and gustatory or hypoglossal nuclei is at hand.

Towards the cephalaic extremity of the vestibular nuclei, the floor of the fourth ventricle becomes very vascular. A rather large vessel is in contact with the nucleus triangularis. This relationship is so close that it would seem almost impossible to change the calibre of this vessel or the pressure within it without affecting the vestibular apparatus. Attention is called to this fact as a possible explanation of the vertigo that frequently accompanies vascular disease.

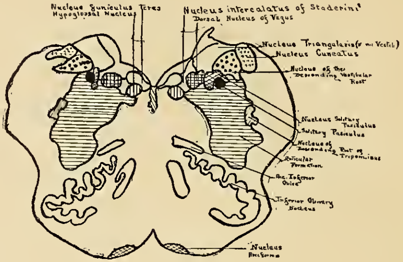

FIG. 5. (SEC. 440)

EXPLANATION OF ILLUSTRATIONS

The terminology used in the accompanying illustrations is that employed by Jacobsohn. However, the following modifications have been made: Taking the entrance of the vestibular nerve as the level at which ascending and descending arms originate, those nuclei which lie cephalad to this level will be considered as termini of the ascending arms and those which lie below this level will be referred to as nuclei of the descending arms.

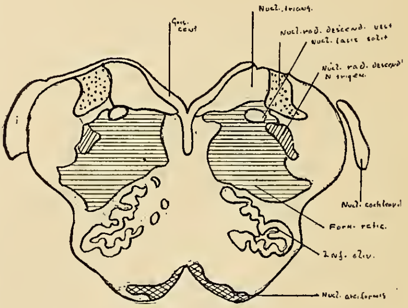

FIG. 6. (SEC. 480)

Fig. 1 shows the entrance of the vestibular nerve, description of which has been made above.

Fig. 2 shows the cephalic pair of vestibular nuclei, description of which has been made above.

Fig. 3 shows the caudal pair of vestibular nuclei, description of which has been made above.

Fig. 4 gives the outlines of the various gray structures of the medulla, at the caudal end of the vestibular nuclei. The close relationship of the vestibular nuclei to the nucleus of the column of Burdach, to the dorsal nucleus of the vagus nerve and to the nucleus of the solitary fasiculus should be noted. From the standpoint of function, in this dorsolateral region of the medulla, are represented from without inwards, the proprioceptive senses of the arm and upper trunk, the more complicated proprioceptive sense of knowledge of position in space, the sense of taste and those enteroceptive senses under the domination of the vagus nerve. Still more internally is the nucleus of the hypoglossal nerve which in terms of function represents the motility of the tongue. Ventral to the vestibular nuclei is the important but ill-understood reticular formation.

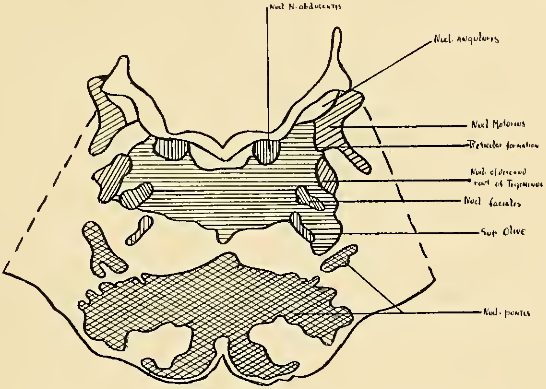

FIG. 7. (SEC. 565)

In Fig. 5 (1 mm. cephalad to Fig. 4) the same structures as in Fig. 4 may be observed, although they have changed in size. The vestibular nuclei have expanded laterally and mesially. The nucleus of the descending root has encroached upon the diminishing nucleus of the column of Burdach, and the triangular nucleus is taking the place of the dorsal nucleus of the vagus. Mesially to the dorsal nucleus of the vagus a new mass has appeared—the nucleus intercalatus of Staderni, which Bárány considers vestibular in function.

Fig. 6 (2 mm. cephalad to Fig. 5) shows that the nucleus triangularis now occupies practically all the floor of the fourth ventricle. The nucleus of the descending root of the vestibular nerve has enlarged and has replaced the area originally occupied by the nucleus of the column of Burdach.

Fig. 7 is 4.25 mm. cephalad to Fig. 6 and 1.25 mm. cephalad to the entrance of the vestibular nerve. The vestibular nuclei occupy the same relative position that they did in the lower figure, but differ from them in that they receive the terminations of the ascending arms. The nucleus angularis (von Bechterew's) is in close relation with the gray matter of the ventricular floor, while the nucleus motorius (Deiters') is imbedded in the white substance of the dorsolateral area of the medulla. Mesial to these nuclei (not shown in the figure) the facial nerve passes ventrolaterally towards its point of exit, and mesial to this is the nucleus of the sixth cranial nerve. Ventral to Deiters' nucleus is the nucleus of the descending root of the trigeminus, which is separated from the facial nucleus, on its ventromesial aspect, by the facial nerve. Ventromesad to the facial nucleus is the superior olive. The functions represented by these structures are, therefore, knowledge of position in space, external rotation of the eye, movements of the face, sensations of the face, and hearing. Due to the vascularity of the ventricular floor in this region—a subject that will be discussed later—hemorrhages with disturbances of the functions mentioned above may easily occur.

Fig. 8 (2.50 mm. cephalad to Fig. 7) shows the same structures and relationships as are seen in Fig. 7.

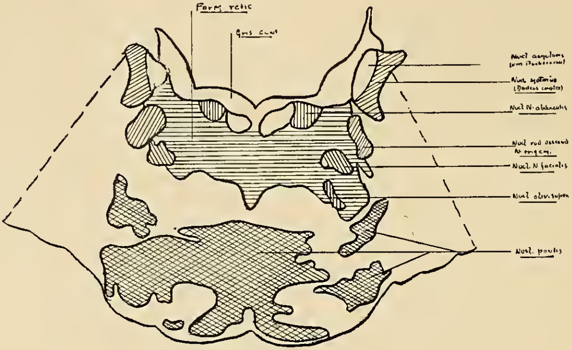
Form. retic
Gris. cent
Nucl. angularis (von Bechterew)
Nucl. Motorius (Deiters complex)
Nucl. N. abducentis
Nucl. rad. descend. N. trigem.
Nucl. N. facialis
Nucl. oliv. super.
Nucl. pontis

Fig. 8. (Sec. 570)

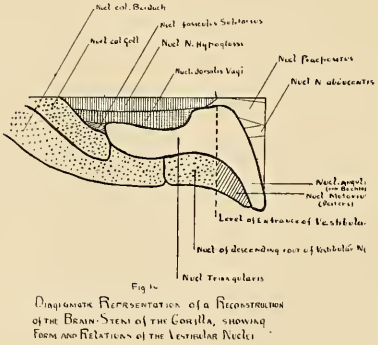
Nucl. col. Burdach
Nucl. fasciculus Solitarius
Nucl. col Goll
Nucl. N. Hypoglossi
Nucl. dorsalis Vagi
Nucl. Paraponties
Nucl. N. abducentis
Nucl. angul. (von Bechterew)
Nucl. Motorius (Deiters)
Level of Entrance of Vestibular
Nucl. of descending root of Vestibular N.
Nucl. Triangularis
Fig. 10
Diagramatic Representation of a Reconstruction of the Brain-Stem of the Gorilla, showing Form and Relations of the Vestibular Nuclei

Fig. 9. Diagrammatic Representation of a Reconstruction of the Brain-stem of the Gorilla, Showing Form and Relations of the Vestibular Nuclei

Fig. 9 is a schematic representation which shows the dorsal aspect of a reconstruction of the nuclear substance of the brain-stem of the gorilla. It reveals the nucleus of the hypoglossal nerve as a cylindrical-shaped body parallel and close to the median line. Laterad to this is another cylindrical-shaped

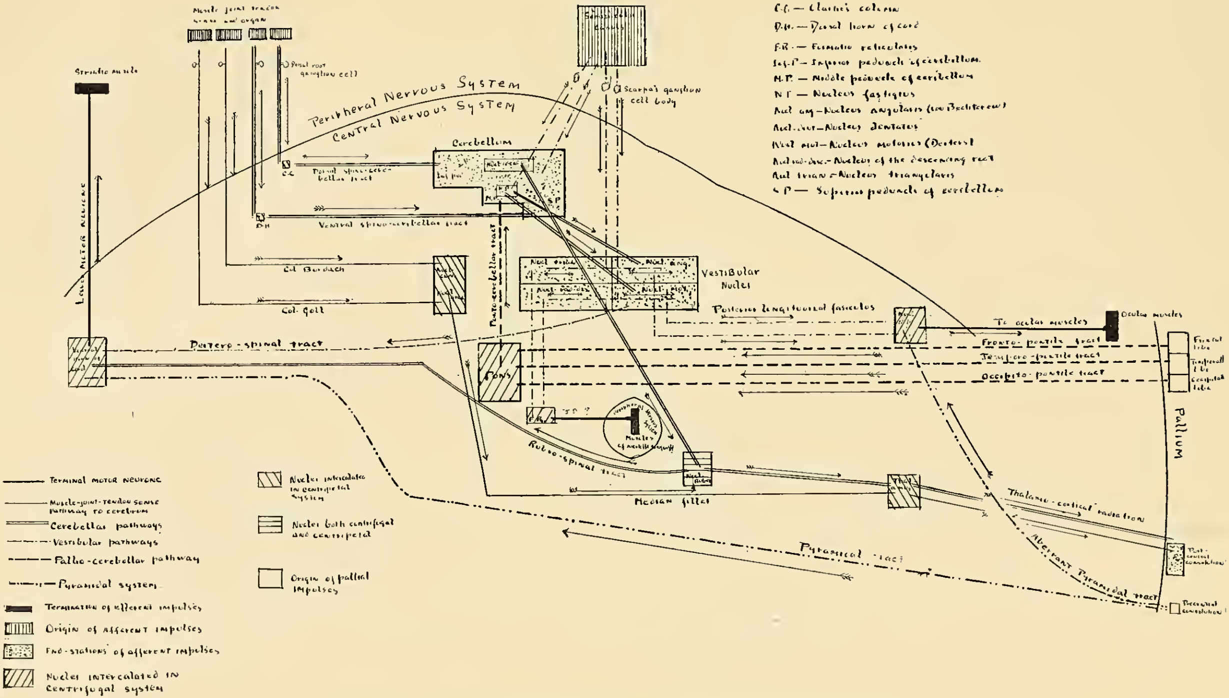
C.C. — Clarke's column
D.H. — Dorsal horn of cord
F.R. — Formatio reticularis
Inf.P. — Inferior peduncle of cerebellum
M.P. — Middle peduncle of cerebellum
N.F. — Nucleus fastigius
Nucl. ang. — Nucleus angularis (von Bechterew)
Nucl. dent. — Nucleus dentatus
Nucl. mot. — Nucleus motorius (Deiters)
Nucl. rad. desc. — Nucleus of the descending root
Nucl. trian. — Nucleus triangularis
S.P. — Superior peduncle of cerebellum
Striatic muscle
Muscle joint tendon sense end organ
Dorsal root ganglion cell
Peripheral Nervous System
Central Nervous System
Semicircular canals
Scarpa's ganglion cell body
Cerebellum
C.C.
D.H.
Dorsal spino-cerebellar tract
Ventral spino-cerebellar tract
Col. Burdach
Col. Goll
Lower motor neurone
Ponto-cerebellar tract
Vestibular Nuclei
Posterior longitudinal fasciculus
To ocular muscles
Ocular muscles
Deitero-spinal tract
Pons
Fronto-pontile tract
Temporo-pontile tract
Occipito-pontile tract
Frontal lobe
Temporal & occipital lobe
Pallium
F.R.
Peripheral Nervous System
Muscles of mastication
Rubro-spinal tract
Median fillet
Thalamo-cortical radiation
Post-central convolution
Aberrant Pyramidal tract
Pyramidal tract
Precentral convolution
Terminal motor neurone
Muscle-joint-tendon sense pathway to cerebrum
Cerebellar pathways
Vestibular pathways
Pallio-cerebellar pathway
Pyramidal system
Termination of efferent impulses
Origin of afferent impulses
End-stations of afferent impulses
Nuclei intercalated in centrifugal system
Nuclei intercalated in centripetal system
Nuclei both centrifugal and centripetal
Origin of pallial impulses

FIG. 10. SCHEMATIC REPRESENTATION OF THE PROPRIOCEPTIVE PATHWAYS AND THEIR RELATIONS AT THE TERMINAL MOTOR NEURONES TO THE VOLUNTARY SYSTEM

nucleus, the dorsal nucleus of the vagus. Soon after the appearance of the nucleus triangularis it expands and replaces the dorsal nucleus of the vagus. Laterad to the triangular nucleus is the upper portion of the nucleus cuneatus, the nucleus of the descending root of the vestibular nerve and the nucleus motorius. Caudad to the triangular nucleus a small portion of the nucleus of the solitary fasciculus is observed. The nucleus of the descending root does not terminate as it appears to in the diagram, but penetrates the nucleus of the column of Goll, in a cone-shaped manner. A dashed line indicates the approximate level of the entrance of the vestibular nerve, and divides the lateral vestibular nucleus into its two components—the nucleus motorius and the nucleus of the descending root. The line of demarcation, however, between the triangular nucleus and the nucleus of von Bechterew, is not so sharply defined. Cephalad to the hypoglossal nucleus is a series of ill-defined nuclei which includes the nucleus præpositus, the paramedian nucleus, the nucleus of the funiculus teres, and the nucleus intercalatus of Staderini. These nuclei at first seem to crowd the nucleus abducentis outwards and downwards, before it occupies its mesial position; it is thus brought into very close relationship with the upper portion of the nucleus triangularis.

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A CASE OF JUVENILE PARALYSIS AGITANS

PRIMARY ATROPHY OF THE PALLIDAL SYSTEM OF THE CORPUS STRIATUM*

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INTRODUCTORY REMARKS

In a recent number of *Brain*,[1] I reported a series of clinical observations and a pathological study of the so-called juvenile paralysis agitans, a type of the disease which is comparatively rare. In the case which was the subject of pathological investigation the disease had existed for twenty-five years, beginning at the early age of fifteen. The only lesion which I was able to discover after a careful examination of the central nervous system was atrophy of the large motor cells of the corpus striatum with some secondary thinning of the strio-hypothalamic radiations—the ansa lenticularis and the ansa peduncularis. The small ganglion cells of the caudate nucleus and the putamen (neostriatal cells) were, in contrast, quite normal in appearance and in number. The essential lesion, therefore, was of the corpus striatum, consisting of a selective atrophy of the large motor cells of the globus pallidus type, a cell type which is characteristic of the globus pallidus but which is also present in the caudate nucleus and putamen. (See Figs. 1 and 2.)

It was my belief that the peculiar motor disturbance of paralysis agitans resulted from the loss of this motor system of the corpus striatum, which I would term the *pallidal system* and which, I believe, controls the automatic and association activities of muscles through connections with important nuclei in the hypothalamic region and the extrapyramidal tracts of the spinal cord (the rubrospinal and other descending tracts). According to this conception, the central nervous system of man has two distinct systems for the regulation of movement: (1) a corticospinal or pyramidal tract system for the control of isolated, synergic movements of cortical origin; and (2) a striospinal or extrapyramidal system for the control of automatic and associated movements.

These two systems may be dissociated by primary neuronal degenerations. Thus, when the pyramidal tract system undergoes primary degeneration, a spastic paralysis would result and degeneration of the pallidal system would produce paralysis agitans. The shaking palsy, according to this conception, is not a definite disease but a syndrome, a special type of central palsy which may be produced by a variety of pathological lesions within the corpus striatum: *e. g.*, primary atrophy, senile degeneration, vascular lesions, tumors, lues, and inflammations.

The juvenile paralysis agitans I therefore regard as a primary system disease, an abiotrophy in the sense in which this term was used by Gowers, and as another example of the vulnerability of motor neurons. In the domain of the corpus striatum and the striospinal system it is the equivalent of the primary degenerations of motor tracts in the corticospinal and spinoneural systems.

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[1] *Brain*, 1917, *Vol.* XL, p. 58

* Presented at the Clinical Conference of the Neurological Department of the College of Physicians and Surgeons, Columbia University, February 25, 1918.

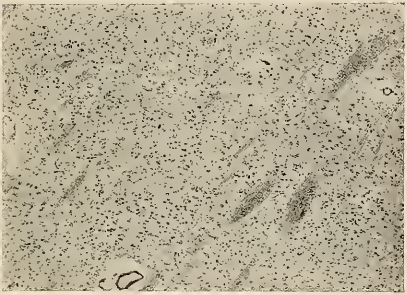

FIG. 1. SECTION OF PUTAMEN, TOLUIDIN BLUE, X 384. FROM JUVENILE PARALYSIS AGITANS OF TWENTY-FIVE YEARS' DURATION

Showing preservation of the small ganglion cells, and almost complete absence of the large motor cells of globus pallidus type. Note the increase of glia nuclei.

Primary atrophy of the pyramidal tract system is followed by the symptoms of spastic paralysis; primary atrophy of the peripheral motor system by progressive muscular atrophy, and primary atrophy of the pallidal system of the corpus striatum by paralysis agitans. The systemic nature of juvenile paralysis agitans receives further confirmation from the not infrequent familial incidence of this disease, thus indicating an inherited biological weakness of certain neurons.

PRESENTATION OF CASE

The patient at the present time is thirty-nine years of age and unmarried. She has been under my observation for about one year, coming to see me at intervals of a month or six weeks. Except for the nervous manifestation, she enjoys very good health.

There is nothing of note in her family history. She is an only child and there is no history of the occurrence of a similar disease in other members of the family. Her mother is still living; her father met with an accidental death some years ago by drowning.

Her previous history reveals nothing of special importance. She passed through the usual diseases of childhood, but none were of especial severity or accompanied by complications. I may say that her internal organs are all normal and that her blood was tested by the Wassermann reaction some years ago and the result was negative.

The malady from which she now suffers began when she was about nineteen years of age. She is unable to recall any definite symptoms before that time. The first symptom was a cramp, or a sensation of cramp, in the right foot. It is always of great value in organic affections to lay stress on the very early symptoms so that, if we meet with a subsequent case in the initial stage, a diagnosis might be more readily made. This cramp in the right foot was not constant; it only appeared when she was tired or toward the end of the day when the muscles felt stiff and rigid. In the morning when she awoke from sleep the cramp had disappeared. This symptom continued for some time and gradually increased in severity so that eventually the evening cramp became a permanent stiffness in the foot and leg.

For several years, or until she was about twenty-three years old, the disability was limited to stiffness and weakness of the right foot and leg. At this time the right hand began to tremble. The tremor of the hand came on very gradually and was not constant, in this respect resembling the initial rigidity of the foot. It appeared at certain times only and then without apparent reason would disappear. Little by little, however, it became a fixture so that in time there was present almost constantly a marked trembling which became more and more marked as the disease progressed. When she was about thirty years of age the first evidence of trouble appeared on the left side. First there was stiffness on moving the leg, and this was followed by a tremor of the hand and arm, a sequence of events not unlike that which had been noted upon the right side. The progress, however, was now much more rapid, probably because the disease was well established, and the degenerative tendency well under way. It is, however, interesting to note that for three years the distribution of the rigidity and tremor was of the hemiplegic type, a not uncommon observation in the juvenile as well as the senile and presenile forms of the disease.

The patient has had no special trouble in masticating or swallowing food. The speech, except for slight tremulousness under excitement, is normal. She has had no pains and no paresthesias. With the exception of the motor disability, namely weakness, rigidity, and tremor, she is quite well and enjoys excellent health. Her mind is perfectly clear, there is no loss of memory, and she is quite cheerful in spite of the chronic and serious nature of her malady.

A few little points are of interest which are sometimes observed in these cases. When there is a sudden and unexpected demand on her activity she will sometimes respond with extraordinary freedom from the usual rigidity and weakness, as, for example, the response to a sudden ring of a doorbell or of a telephone bell. On these occasions she will rise and pass to the door with unaccustomed freedom of movement in contrast to the same act performed in response to a more conscious effort. She also runs much better than she walks. After a good night's rest there is often an improvement in the rigidity and tremor for twenty minutes or half an hour, but rarely longer, and then the accustomed symptoms supervene. Since the onset of the disease the symptoms have never disappeared for any longer period of time.

I may say that no symptoms which characterize other organic diseases of the central nervous system have ever been noted. She has never experienced amblyopia, diplopia, or vertigo; there have been no sphincter troubles, no lancinating pain, no paresthesias or ataxic disturbances. The clinical picture has been limited to a weakness, rigidity, and tremor, for many years hemilateral, subsequently assuming a bilateral distribution and characterized by very gradual progression. Unusual was the early age of onset of the disease.

(Dr. Hunt proceeds to the demonstration of the case.)

Examination.—You can readily observe the marked trembling of the hands and arms; this is also present in the legs, and in the body. There are four or five vibrations to the minute. It is interesting to note that the tremor is in the same tempo in all of the affected parts. The tremor of the right hand is coarser and has a greater amplitude than that of the left and is more marked in the wrist and arms than in the fingers. It is increased by movement and by excitement, but is not of the intention type. As the finger is carried to the tip of the nose the tremor increases in intensity during the passage of the movement, but as the finger tip reaches the nose the extremity remains fairly quiet in contradistinction to the tremor of multiple sclerosis. In the latter condition, on attempting the finger-nose test, the finger cannot be kept on the face, the hand flying off wildly every time the effort is made. On the contrary, this patient's tremor diminishes, in this test, as the finger approaches the nose.

Fig. 2. Section of Globus Pallidus, Toluidin Blue, x 384. From Juvenile Paralysis Agitans of Twenty-Five Years' Duration

Showing general atrophy and diminution in number of the large motor cells of the globus pallidus. There is moderate increase of the glia nuclei and numerous large hyaline concrements staining intensely blue.

The handwriting also shows very definitely the evidence of tremor. She states that she is able to control the tremor much better while kneeling on the floor. (See Fig. 4.)

There is no tremor of the tongue, face, or eyelids. There is no nystagmus. The speech is perfectly clear, although tremulous and at times a little slow. The face does not show the typical Parkinson mask. It is, however, a little stiff and there is slight fixity of expression, as you will observe when I ask her to smile. The voluntary innervation of the facial muscles is very good; she shows the teeth, closes the eyes, and wrinkles the forehead normally on the two sides, so that the slight facial immobility is one of emotional expression and not of voluntary movement.

In the muscles of the neck there is a tendency to rigidity which is a little greater on the right side. You will note that both sternomastoid muscles stand out quite prominently and their contour is well defined. (See Fig. 3.)

In testing the right hand and arm there is distinct resistance to passive movements. This is a condition of marked rigidity of the musculature. The muscles are firm and of a waxlike consistency, and there is on passive movement a distinct, rhythmical interruption, the so-called cogwheel symptom due to the intrinsic tremor tendency of the muscle. This is present also on the left side, and in the legs as well.

The reflexes are as follows:

Pupils.—Equal on both sides and respond normally to light and accommodation. The sympathetic reflex is also present.

Skin.—All normal. The abdominals and epigastrics are normal and equal on the two sides.

Plantar.—Of the normal type. There is no Babinski response. The tendon reflexes of the arms are normal and equal on the two sides.

Knee Jerks.—Normal.

Achilles.—Active and equal on the two sides.

There is no true clonus either of the patella or of the ankle. At times, however, owing to the tremor tendency, a pseudoclonus may be induced which, nevertheless, is exhaustible and is dependent upon the tremor and lacks the characteristics of true ankle clonus (a fact which is not unimportant as the juvenile paralysis agitans in its early stage is not infrequently regarded as multiple sclerosis). As there is no clonus, no Babinski response, and no loss of the abdominal reflexes, a disease of the pyramidal tract system may be definitely excluded.

The sensory tests are all normal. There are no disturbances whatever of superficial or of the deep sensibility.

There is no disorder of the bladder or rectal functions.

So much for the general neurological picture.

There are, however, certain features to which I would direct your attention. A most striking symptom is the fact that in walking the patient's arms do not participate. There are not the rhythmical alternating movements of the arms which are normally presented in

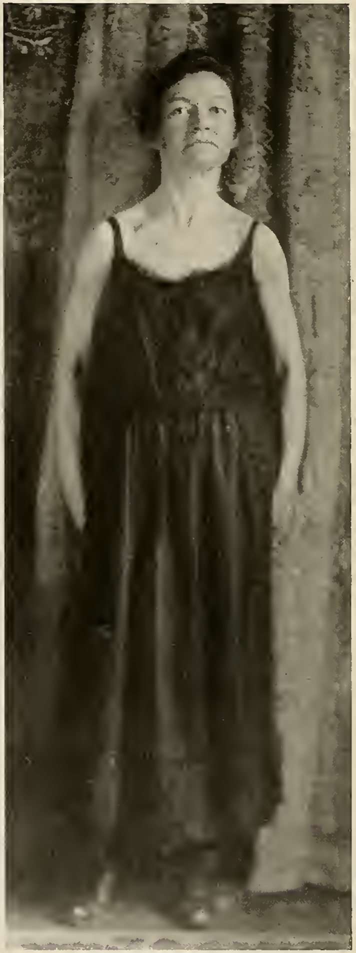

Fig. 3. Showing Attitude and Facial Expression

walking and running. This rhythmical swing of the arms is a vestigial remnant of the quadruped gait in man, as was pointed out by Dupré some years ago, and will be found to have a special significance when we come to the interpretation of the clinical picture. This associated movement of the arms is of great assistance in walking, therefore this patient has a distinct disability in walking, for, no matter how fast she goes, her arms are motionless. Under my direction she has been taking lessons for a year in the art of moving her arms when she walks, yet she cannot acquire this swing which is readily and unconsciously performed by all of us. She makes a conscious and very evident effort, but it seems impossible for her to acquire this very simple movement which is so natural to all. It would be comparatively easy to teach her to perform difficult movements which are acquired and are of cortical origin. For example, when she takes off her coat, the act is performed as you or I would do it. Such an action has been acquired and is of cortical origin. Yet she has lost and is unable to acquire the simple movement of swinging her arms in walking, showing that the automatic and association activities of movement are those which are essentially involved.

You will also observe that as she attempts to sit down, the patient walks up to the chair, turns around, and falls back into the chair, almost losing her balance. She has lost the power to utilize certain normal associated movements which accompany this act, such as placing her feet under the chair and using her hands and trunk muscles to maintain the proper balance. When a blanket is placed beneath her and she is asked to lie down on the floor, she falls back and her legs rise helplessly in the air. It is impossible for her to learn to assume the recumbent posture in the normal manner. Something is gone which has to do with the association mechanism for the control of automatic and associated movements.

(Dr. Hunt offers his hand to the patient and tells her to pull herself up, while sitting in the chair.) She takes hold of my hand, but makes no traction or pulling effort. The grasp is weak and there is no pull. She rises but without the assistance which my hand would give her. She has lost the important mechanism which controls and regulates such movements.

You will observe another interesting symptom and one which when present is quite characteristic. This is the absence of a quick extensor movement of the hand on making a fist. Instead, her fingers are simply flexed in the palm without the slight dorsal kick which usually accompanies the act. It is of interest to note, in this connection, that the extensor kick is exaggerated in pyramidal tract lesions and is known as Strümpell's radial phenomenon. Another peculiarity is the absence of strong abduction of the thumb when the hand is opened. You will observe that the fingers are extended somewhat slowly but without that spreading movement of the thumb and fingers which characterizes the normal act.

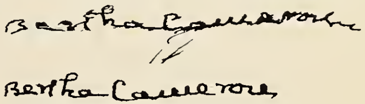

Fig. 4. Specimens of Chirography

Upper line written while standing. Lower line written while kneeling.

There are also other disturbances of association of movement. When the arm is elevated to the horizontal, there is not the usual associated contraction of the erector spinæ group on the opposite side, and in walking ,you will observe, the feet are shuffled along the floor and there is not the normal combined movement of flexion of thigh, knee, and ankle which characterizes the normal step. The leg is lifted, but there is not the simultaneous flexion of the foot.

COMMENTS

The case which I have just demonstrated presents the three characteristic symptoms of paralysis agitans, namely, a paralysis of certain types of movements which may be summed up as automatic and associated movements; a general rigidity of the musculature; and a spontaneous, rhythmical tremor. The affection has been slowly but steadily progressive over a number of years and is peculiar in the rather youthful age of onset. I have had the opportunity to study several cases of this juvenile type, one beginning at the very early age of thirteen years. In the early stages of the affection, especially when the tremor is conspicuous and of great amplitude, there is a certain resemblance to multiple sclerosis, which is enhanced by the occasional presence of a tremor of the eyes and a pseudo-ankle clonus of tremor origin. The absence, however, of a true intention tremor and all signs of pyramidal tract disease will usually serve to exclude this affection.

Another disease which bears a certain resemblance to this condition and which has awakened considerable interest in recent years is the progressive lenticular degeneration described by Kinnier Wilson. This affection, however, has a more rapid course and is associated with fever and great emaciation. Especially characteristic, for the differential diagnosis, is the presence of choreo-athetoid movements and mobile spasms which are entirely lacking in paralysis agitans.

The essential pathological lesion underlying this disorder is, according to my conception, a primary atrophy of the efferent motor system of the corpus striatum, causing a break in the striospinal mechanism which controls automatic and associated movements. This mechanism is infracortical, and such activities are, therefore, unconscious; it is, however, under the control of the higher cortical centers, so that a certain amount of voluntary control exists.

The rigidity I would explain by a loss of the inhibitory influence of the corpus striatum upon the lower spinal centers, in the same manner as the spastic hypertonus results when the cortical control is lost after lesions of the pyramidal tracts. A spastic hypertonus, therefore, results from loss of cortical inhibition and the rigid hypertonus of paralysis agitans from loss of striatal inhibition.

The tremor is rather a puzzling symptom and is not so readily explained. Hughlings Jackson introduced the interesting hypothesis that tremor was related to rigidity and considered the two of similar nature and origin. As he expressed it, "Tremor is rigidity spread thin, and rigidity is tremor run together." While there is much in this theory that is suggestive, it fails to explain all the phenomena of the disease. A considerable percentage of paralysis agitans cases run their course without tremor, although rigidity is present, and cannot be distinguished from those cases in which tremor and rigidity are associated.

In recent years, evidence has accumulated to show that the voluntary muscular system is under the control of a dual innervation: (1) a medullated nerve fiber and a motor end plate which is associated with the anisotropic disc system; and (2) a non-medullated nerve, a sympathetic end plate, and the sarcoplasmic substance of muscle. I have elsewhere suggested that these two muscle systems are under the control of special correlating centers which in turn are under the control of the corpus striatum. In the case of the sarcoplasmic system a rigidity would result if the striospinal inhibition were removed; while removal of the inhibition from the anisotropic disc system would allow a spontaneous tremor to develop.

I recognize that I am wandering into a hypothetical field in postulating special correlating centers for the dual systems of striated muscles. It may, however, assist in stimulating interest and speculation in a still obscure field of neural physiology.

WILSON'S DISEASE—PROGRESSIVE LENTICULAR DEGENERATION*

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The following case of progressive lenticular degeneration is presented as a preliminary report to a further and more extended study of the motor disturbances consequent upon lesions in the corpus striatum. The chief pathological characteristics of Wilson's disease are a marked cirrhosis of the liver beginning in youth or adolescence and accompanied by a simultaneous degeneration in both lenticular nuclei. Since the brilliant and comprehensive work of Kinnier Wilson describing the syndrome which now bears his name, a number of attempts have been made to still further elucidate the conception of progressive lenticular degeneration. Explanation of the simultaneous hepatic and cerebral changes is still being sought. Furthermore, the symptom-complex resulting from this particular brain degeneration has done much to stimulate a renewed interest concerning the nature and function of the corpus striatum, that part of the end-brain which remains as fascinating to the investigator as it is perplexing to the clinician.

The earliest reported case of this rare and interesting disease was described by Frerichs in 1854 in a treatise upon the diseases of the liver. Frerich's patient was a boy of ten years of age, normal in all respects, with a history of having developed dysarthria, dysphagia, weakness of the limbs and tremors, while later, emaciation, slowness of muscular movements without paralysis in the strict sense, and a loss of facial expression made their appearance. There were no symptoms of involvement of the liver during life and sensibility remained intact. Extreme cirrhosis of the liver was the sole pathological finding at autopsy. Although the symptom-complex had all the unmistakable signs which go to make up Wilson's disease as it is at present recognized, Frerichs himself did not discern the true nature of the disturbance and, in fact, made no observation concerning the state of the corpus striatum. No observation of this symptom-complex is found in the literature until thirty-four years later. Gowers in 1888 published in his manual notes of a case which he called "tetanoid chorea." He recognized a combination of nervous symptoms which was entirely unlike that of any other nervous disease, but he failed to attach any importance to the changes in the liver. In fact, in his first published notes of this case, he omitted all mention of the cirrhosis and it was not until a later search of the records of the National Hospital was made, many years afterward, that he was struck by the fact that the extrapyramidal tract disease was associated with a well-marked cirrhosis of the liver in a young subject. Gowers' first patient was a boy of ten years of age. A short time after his first publication, a sister of this patient, a girl of fifteen years, came under his observation, suffering from identical symptoms.

In 1906, Gowers[1] reviewed the records of these two cases and published a paper on "Tetanoid Chorea and its Association with Cirrhosis of the Liver." Clinically and pathologically, these three cases showed the closest resemblance, namely, (1) grave and progressive nervous symptoms; (2) no obvious lesion of the brain or cord; (3) a latent and unsuspected hepatic cirrhosis. In 1890 Omerod published in the *Saint Bartholomew's Hospital Reports* notes of a similar case. Homén of Helsingfals in 1890 and then more fully in 1892 reported three cases occurring in the same family, two brothers and a sister. Homén noted bilateral lenticular softening in all of his cases, but just as Gowers had regarded the cirrhosis of the liver as merely an incident in the disease of the nervous system, so Homén failed to attribute any of the symptoms to the changes in the basal ganglia, but thought that what he termed "cortical irritation" and "general intellectual deterioration" explained the clinical condition.

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[1] *Rev. Neurol. & Psychiat.*, 1906.

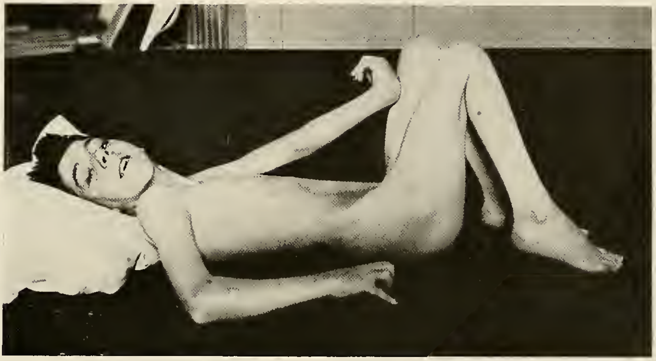
* Presented at the Clinical Conference of the Neurological Department of the College of Physicians and Surgeons.

Wilson in 1912 published his personal observations on four cases, together with an exhaustive study of the previously reported cases presenting the characteristic symptom-complex. Since the publication of this monograph, a number of cases have been reported in England and France, and in this country.

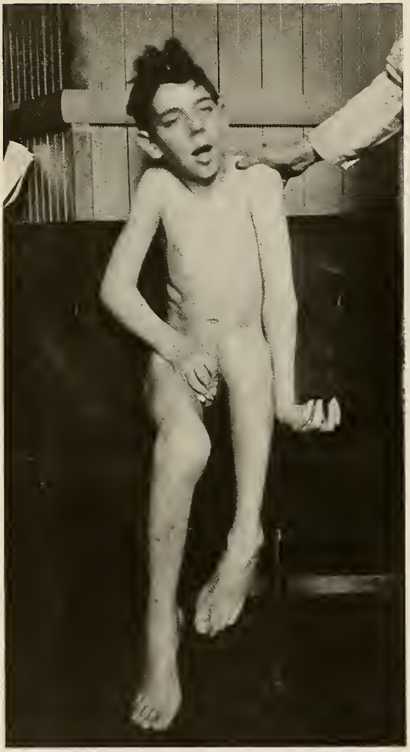
Case No. 13,889, P. H. H. McK.—A Scotch boy, fifteen years of age, was admitted to the Presbyterian Hospital, having the complaint of stiffness of the right arm and difficulty in speaking. Upon attempting to use it for any purpose whatsoever, the right hand and arm trembled. The boy was born in Scotland, but came to the United States when he was four years old. Since that time he has lived in New York City, except for nine months during the year 1913, when he was in Canada. Until his present illness, he was considered a healthy and normal boy. He never failed to be promoted with his class at school and graduated successfully from grammar school. At the age of two years he had measles, and scarlet fever when he was eight years old. He made a good recovery from both of these illnesses. At eight years of age he had a tapeworm. When he was ten years old he had his tonsils and adenoids removed, because of some difficulty in breathing. He has not been subject to sore throat or to any other illnesses. His previous habits and general physiological status were good. He did not use tea, coffee, alcohol or tobacco. His sexual functions were normal; he slept well; his appetite was good; his bowels regular, and he showed the normal increment in weight during his development.

Family History.—His mother and father are living and in good health. He has two brothers and a sister younger than himself; all of them in good health. There is no history of any similar illness in the family of either parent, nor, in fact, is there any record or recollection of familial nervousness or neurotic tendency. There is no history of syphilis, deafness, blindness, alcoholism, or feeble-mindedness in the direct or collateral branches of the patient's family.

Present Illness.—In August, 1912, without any known cause, the patient began to notice a change in the control of his right hand. When he tried to write or button his clothes the fingers of his right hand and forearm would shake stiffly. When he was excited he found that both hands shook in a stiff and rigid manner. In the four months following he was massaged regularly and seemed to improve a little, but this trouble was still present and annoying. In the hope that a change of climate might improve his condition, he went to Canada in December, 1912. He remained there during the following nine months although there was no perceptible change for the better in his trouble, and those who were watching him felt that he had grown worse. In August, 1913, about seven weeks before his admission to the Presbyterian Hospital, his aunt with whom he was living heard him breathing very heavily one night and on going in to see if she could help him, found that he did not recognize her and seemed to be in a dazed condition for some moments. In the morning the pillowcase was stained and the patient's family concluded that he had had a convulsion during the night. The patient himself stated that his tongue was sore after this occurrence. It is certain from a careful review of his history that the patient had no other general or localized convulsive attacks and consequently, in the absence of corroborative evidence, this alleged convulsion is open to some question. It is equally certain, however, that since the night upon which he had this peculiar seizure, his right arm had grown stiffer and the tremor in it increased. For a number of years it has been characteristic of him to keep his mouth open in the manner often observed in younger children, but his mother feels certain that during the seven weeks prior to his entrance into the hospital his mouth was held more constantly and more widely opened, while his entire face became more expressionless and his speech more difficult to understand. At times during his stay at the hospital, the left arm became temporarily stiff and on several occasions he had cramp-like pains in his right arm. In the early part of December, 1913, his legs grew stiff so that it was no longer possible for him to move about and he became confined to his bed from this time. This same type of stiffness made its appearance in his entire body musculature, so that his neck and back became rigid, the muscles of his face tense and drawn, and apparently, to some degree, at least, the same condition existed in his tongue. This muscular rigidity brought on certain malattitudes in his legs and in his arms. He would lie on one side or the other with his thighs flexed upon the pelvis, his legs flexed upon the thighs, with the feet in plantar flexion and inversion. His arms were also held in contracture-like attitudes; the left arm was internally rotated, the forearm extended on the arm, the wrist flexed upon the forearm, and the fingers, particularly the index finger and thumb, flexed at the proximal joints. The right arm was internally rotated, slightly abducted, and the forearm flexed upon the arm; the wrist was flexed upon the forearm, as were also the fingers, although to a less degree than on the left side. From this hypertonic condition of his muscular system, it came about that the patient could, with difficulty, maintain the sitting posture, since the slightest push would cause him to fall over backward, if not supported. In falling, his limbs would maintain the original attitude assumed before he began to fall backward, so that his whole body seemed to move as a single piece. When standing, the same stiffness in the muscles was apparent. With support he could stand upon his toes and even take a few steps. After a few moments in this position, locomotion improved to such an extent that he could move forward with slight assistance. The tremor referred to as first appearing in his right hand and forearm soon spread to all of the muscles of the right upper extremity, and in the early part of December, 1913, a similar tremor was present in the left hand. The characteristics of this tremor were in many respects similar to those of paralysis agitans; it made itself manifest in the small muscles of the hand, in the forearm and arm. It was a tremor which was monomorphous, having the same rate and rhythm, although during motion of the arm the amplitude of the tremor, as well as its force, increased considerably. In this respect, it showed a notable difference from the tremor of paralysis agitans. Emotional disturbances also increased the force and amplitude of the tremor.

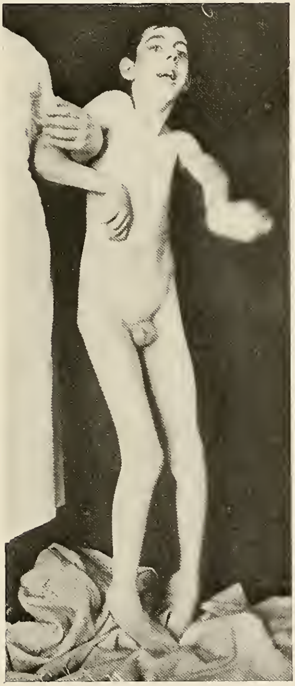


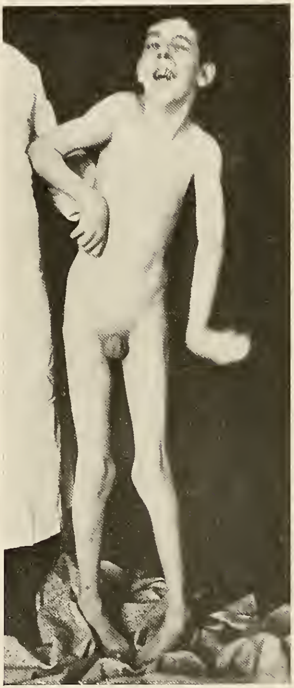
In spite of the increasing hypertonicity of the muscles and the increasing tremor in the upper extremities, there was a remarkable degree of voluntary control over the muscles, and it was difficult, at all times, to detect any real paralysis in the parts affected. He could move his legs and arms at will, but his efforts in these respects gave the impression of a voluntary exertion, laboring in the performance of an act against the resistance of his muscular rigidity. Tremor and hypertonicity alike increased rapidly as the disease progressed. The patient began to experience some difficulty in swallowing in the early part of December and his speech became still more difficult to understand. From time to time he would give voice to a peculiar cry which did not seem particularly expressive of pleasure or pain nor did it have any apparent provocation in his surroundings. When questioned concerning this he could give no satisfactory explanation of its significance and seemed, in fact, to be quite unconscious of its occurrence. He suffered from no pain other than the occasional cramplike attacks in his extremities. His intellectual faculties remained keen and although his facial expression was suggestive of imbecility, it required but a moment's conversation with the patient to convince one that his mind was alert, that he was well orientated in all respects, and far from being depressed by his own distressing condition, he retained a certain sense of native humor even to the time when the toxic effects of his disease caused his mind to wander. His appetite was good, his bowels regular, he showed no evidence of biliary or other pigmentation in his skin, conjunctiva or mucosa. Micturition was normal except for a short period before admission to the hospital when he had some difficulty in passing water. His temperature upon admission was 99 4/10, his pulse 110. With slight elevations, pulse and temperature remained at this level until December 13, when his pulse increased to 140, and with this there was an increase of his respiration to 66. The temperature also began to rise and for the next week maintained an average height of 102, the pulse becoming very irregular and unusually rapid. During this time he emaciated to a considerable degree, suffered from profuse sweats, and gave all the appearances of a severe toxemia. On January 2, 1914, his temperature rose to 104, the next day to 105, and on January 4, 1914, he died.



Since the appearance of his first symptoms there was never a time when he was free from tremor and stiffness in his right arm, that is to say, his disease was unintermittently progressive from the time of the onset.

General Physical Condition.—Upon examination, he appeared to be a well-nourished boy, rather small for his years. Pubic and axillary hairs scanty. Tegumentary system normal. There was no abnormal pigmentation, cutaneous or mucosal, dependent upon or independent of vascularization. There was a marked degree of dermatographia over the entire body. Sweat secretion on admission was normal, later it became excessive. There were no unusual markings or other surface peculiarities. His glandular status, including thyroid, thymus and lymph glands, liver and spleen, was normal. His cardiovascular condition showed a regular pulse, rate 110, systolic 115, diastolic 80. There were no murmurs in any of his cardiac valves. Respiration was normal. Gastro-intestinal and genito-urinary systems were normal.





NEUROLOGICAL EXAMINATION. *Abnormal Attitudes and Deformities.*—The upper extremities were held more or less rigidly fixed in the attitudes already described and illustrated in the accompanying photographs. The same is true of the lower extremities, of the head and back.

Gait.—Because of the muscular rigidity, the gait became progressively more defective, but for the first three weeks of his residence in the hospital the patient was able to get about with some difficulty and without much assistance; later locomotion became impossible. If he walked the gait was digitigrade.

Coördination.—In the early phases of the disease there was no definite disturbance, either equilibratory or nonequilibratory, but subsequently, because of the muscular rigidity, it was difficult to estimate the state of his coördination. In consequence, it is impossible to make any valid statements concerning his diadochokinesis and synergism.

Skilled Acts.—Although there was no definite paralysis, there was, on the other hand, a definite interference with the control of his volitional movements, occasioned by a marked stiffness in the muscles. No apraxia, no dyspraxia, no aphasia. The patient, however, did suffer from a pronounced dysarthria which was attributed to the hypertonus in the muscles of the jaw, lips, tongue and pharynx.

Reflexes:

(*a*) *Deep:*	*Right*	*Left*
Jaw	2	2
Pectoral	2	2
Biceps	2	2
Triceps	2	2
Radial	2	2
Ulnar	2	2
Wrist	2	2
Scapular	2	2
Suprapatellar	2	2
Patellar	2	2
Achilles	2	2
Dorso-cuboidal, exten.	2	2
" flex. (Mendel-Bechterew)	2	2
Periosteal (shin) uncrossed		
" crossed, R. to L.	L. to R.	

(*b*) *Superficial:*	*Right*	*Left*
Ciliospinal	1	1
Supra-umbilical	1	1
Supra-pubic	1	1
Upper lat. abdom.	1	1
Lower lat. abdom.	1	1
Cremasteric	1	1
Bulbo-cavernosal		
Anal		
Plantar flexion	1	1
Babinski	0	0
Chaddock	0	0
Oppenheim	0	0
Gordon	0	0
Schäfer	0	0

Abnormal Involuntary Movements.—A tremor was more or less constantly present in both upper extremities, most marked, however, in the hands, the rate was 5–6 per second, it was rhythmical and of relatively small amplitude; it was increased by action, not only of the arm but by voluntary movements of the legs, of the head or the trunk. The tremor disappeared during sleep, was increased by emotional influences, as well as by attempts at the performance of any act carried on by the upper extremities. There were no twitching, choreiform movements, athetoses, or spasms, and with a possible exception of a questionable convulsion reported by the mother, no convulsive seizures.

Muscle Strength.—The muscular strength upon admission to the hospital was apparently normal and under the control of the will. Toward the end of the disease and especially with the appearance of the increasing toxicity, there was a marked impairment of muscle power.

Muscle Status.—There was no change in the volume of the muscles, although their consistency seemed to increase, due to rigidity. There was a marked increase of muscle tone in the entire somatic musculature and also in the jaw muscles and muscles of expression. The electrical reactions were normal.

Myotatic Irritability.—The myotatic irritability of the muscles was increased.

AbnormalAssociated Movements.—Such tests for these abnormal phenomena as were made elicited none of the well-known signs.

NERVE STATUS.—The nerve status was normal.

GENERAL SENSORY EXAMINATION.—Both phases of sensibility were normal, the only subjective disturbance being an occasional painful cramp in the extremities. All qualities of somesthetic sensibility were normal, including touch, temperature, vibratory, pressure, muscle-tendon, and pain. There was no astereognosis and no barognosis.

CRANIAL NERVES. *Olfactory Nerve and Nose.*—Normal.

Optic and Oculomotor Apparatus.—Normal. Visual acuity was normal; the eye grounds normal; reaction of the pupils to light, accommodation and convergence, normal. No exophthalmos, ptosis, strabismus or nystagmus.

Acoustic Nerve and Ear.—Both divisions of the eighth nerve gave normal responses to all tests.

Trigeminus Nerve and Mouth.—There was no loss of motor control and no sensory disturbance. The muscles of the jaw presented the same degree of hypertonicity.

Facial Nerve and Face.—There was no palsy in either side of the face, although the facial muscles were rigid and gave the mouth a gaping appearance. There were no disturbances of taste on the tongue.

Glossopharyngeus, Vagus, Pharynx and Larynx.—The patient suffered from a slight degree of dysphonia and a progressive dysphasia.

Spinal Accessory Nerve and Neck Muscles.—There was no paralysis in this area of innervation, but the neck muscles were hypertonic.

Hypoglossal Nerve and Tongue.—No paralysis of the tongue, although the intrinsic musculature was hypertonic.

Laboratory Tests.—The blood examinations are as follows:

BLOOD EXAMINATIONS

Date	*W. B. C.*	*Polys.*	*Lymphos*	*Eosinos*	*Large Monos.*
Nov. 13	7.200	73	23	4	
Nov. 21	10.600	88	70		2
Nov. 24	12.600	71	26	3	
Jan 2	13.800	68	32		

Blood Wassermann negative to alcoholic and cholesterin antigens.

Spinal fluid showed no increase in globulin, five cells. Wassermann negative in all dilutions.

Urine negative. Stool negative for parasites and ova.

Carbohydrate assimilation limits were as follows:

Nov. 21. 100 gms. glucose at 8 a. m. on fasting stomach. Patient was involuntary and only one specimen was obtained. This was at 10 a. m., and amounted to 18 cc. It contained glucose 1.19%.

Dec. 1. 50 gms. glucose at 8 a. m. on fasting stomach. Involuntary. No specimen obtained till 2 p. m., when a specimen containing .184% was obtained.

Dec. 12. 50 gms. glucose at 8 a. m. on fasting stomach.
Specimen at 10 a. m. = No glucose
Specimen at 12 noon = No glucose
Specimen at 2 p. m. = glucose .023 gm.

Dec. 15. 50 gms. levulose at 8 a. m., on fasting stomach.
Specimen at 12 noon = 60 cc. Levulose .897%
Specimen at 2 p. m. = 125 cc. Levulose .368%. (Total, .46 gms.)

POSTMORTEM AND PATHOLOGICAL REPORT.—The body is that of an emaciated boy about fifteen years old. Rigor mortis is not present. The body is still warm. The body is turned upon its left side and both legs are strongly flexed upon the abdomen. The left leg is underneath the right leg. The right leg lies at an angle of about 45° to the thorax. The legs are bent up upon the thighs. The left arm is extended, the wrist bent to an angle of about 45° and the fist is clinched. The right forearm is bent at the elbow and extends across the abdomen. The wrist is flexible, fist is closed. The lower jaw is dropped down leaving the mouth gaping. Teeth are in good condition. The skin is slightly eroded over the right anterior superior spine of the ilium and over the inner surface of the right knee. The feet are extended downward. The skin of the back is covered with numerous small whitish papules which are covered by a whitish scale. The inguinal lymph nodes are palpable on both sides.

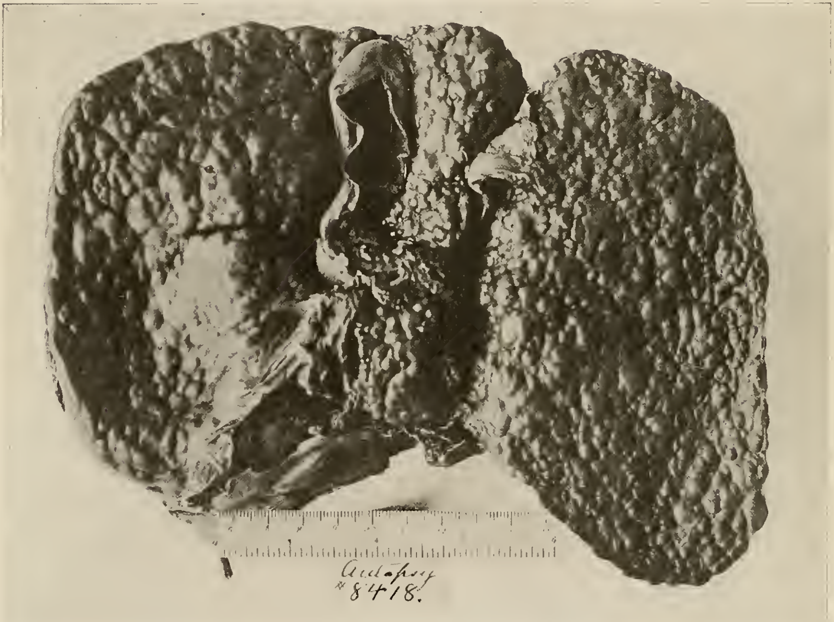
On opening the abdomen the organs are found normally disposed. A hobnail liver is seen extending below the costal border 4 cm. in the midline. There is no free fluid in the peritoneal cavity, and the peritoneum is everywhere smooth and glistening. The appendix is retrocecal. It curves upon itself in an *S* shape and measures 6 cm. in length. The bladder is distended. The lymph nodes in the mesentery of the large intestine are enlarged, averaging 1 cm. in diameter, and are dark purplish red. Although most of them are soft, three or four are firmly calcified. The colon is comparatively large. The diaphragm extends to the sixth space on the left and the fifth space on the right.

On opening the thorax the organs are found normally disposed. Apparently the *thymus* has been entirely replaced by fibrous tissue. There is no fluid in either pleural cavity and there is no excess of pericardial fluid.

Left Lung.—The left lung weighs 200 grams. The pleura is everywhere smooth and glistening. For the most part, the lung appears light reddish pink in color, and numerous small emphysematous blebs are seen. In the lower lobe there are some dark elevated areas which feel moderately firm. The diaphragmatic surface is quite dark, in places almost black. There are some small pin point hemorrhages in the pleura. Throughout the lower lobe a number of hard nodular areas are felt. On section the upper lobe in general is light red in color and appears normal. Around the root of the lung there are a number of darker granular elevated areas which are confluent bronchopneumonia. The lower lobe shows numerous dark granular areas of confluent bronchopneumonia.

Right Lung.—The right lung weighs 200 grams. The pleura is everywhere smooth and glistening. The pleura is light pink, almost white in color, showing many emphysematous blebs, except for that covering the lower half of the upper lobe which is mottled by dark red areas. On section the lung appears entirely normal except for the lower half of the upper lobe which is occupied by confluent areas of bronchopneumonia. From the center of some of the elevated dark granular areas small droplets of pus can be expressed. There is also a small patch of bronchopneumonia in the apex. The bronchi are filled with frothy mucus, the pulmonary vessels are normal. The bronchial lymph nodes average 1 cm. in diameter and are deeply pigmented.

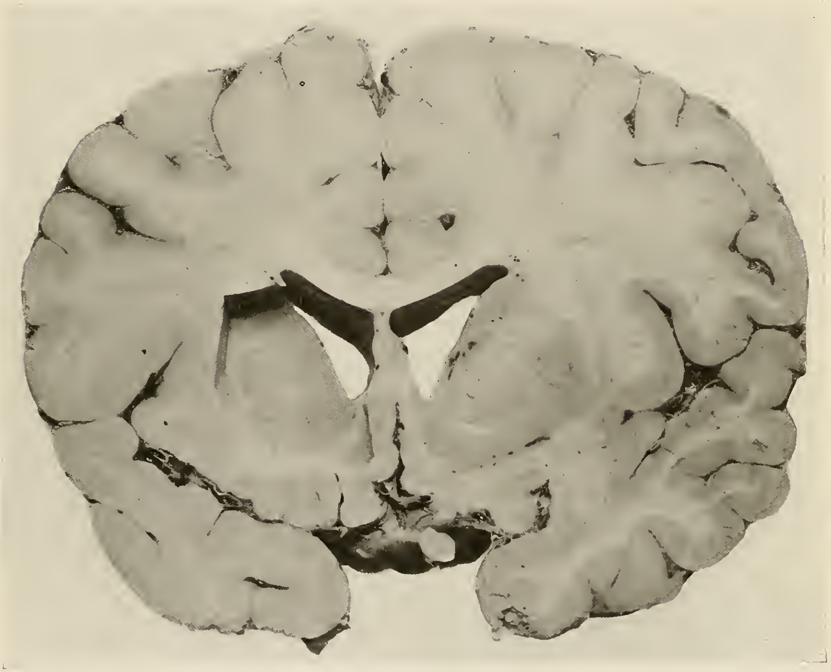
Heart.—The heart weighs 200 grams. The pericardium is everywhere smooth and glistening. The foramen ovale is closed. The *tricuspid orifice* measures 10 cm. in circumference and the valve is normal. The *pulmonary ring* measures 6½ cm. in circumference and the cusps are delicate. The *ventricular wall* averages 4 mm. in thickness. The endocardium lining the left ventricle is slightly thickened by whitish fibrous tissue. The *mitral orifice* measures 7 cm. in circumference. The edge of the aortic cusp of the mitral valve is thickened. There are no fresh vegetations. The papillary muscles are short and stubby. The endocardium lining the ventricle is slightly



Showing Cirrhotic Liver

thickened. The *aortic orifice* measures 5½ cm. in circumference, and the valves are normal. The *ventricular wall* averages 1½ cm. in thickness and the *ventricle* measures 6 cm. in length. The coronary arteries are normal.

light brownish red in color and the depressions are dark purplish red. On section the demarcations of the lobulations are clearly seen. The liver tissue appears light brownish in color. The lobules are indistinctly visible.



Showing Spongy Condition of Putamen

Spleen.—The spleen weighs 175 grams. The surface of the spleen is smooth and a dark slate gray in color. On section the spleen appears dark purplish red in color. It is moderately firm and the Malpighian corpuscles are beautifully seen as large white dots against the dark splenic pulp. The connective tissue is prominent.

Liver.—The liver presents a most classic picture of atrophic cirrhosis. The liver weighs 1000 grams. The whole surface of the liver is covered by irregularly sized rounded projecting lobules producing a most beautiful hobnail effect. The projecting portions are

Gall-Bladder.—A thin mucoid bile can be expressed into the duodenum through the papilla of Vater. The gall-bladder appears normal.

Kidneys.—The kidneys are alike in size and appearance. Each weighs 150 grams and measures 10 x 3½ x 4½ cm. The capsule strips with ease leaving a smooth surface. The stellate veins are indistinctly seen as the kidney is quite congested. On section the cortex is found to average ¾ cm. The cortex and medulla are congested, the cortex being dark purplish red in color and the medulla slightly darker. The striations are straight and easily seen. The glomeruli can just be made out. The pelvis is normal. Both ureters are dilated averaging about ¾ cm. in diameter.

Urinary Bladder.—The bladder is distended with urine and extends up 5 cm. above the symphysis. A probe can be passed through both ureters into the bladder with no resistance.

Prostate.—The prostate is not hypertrophied.

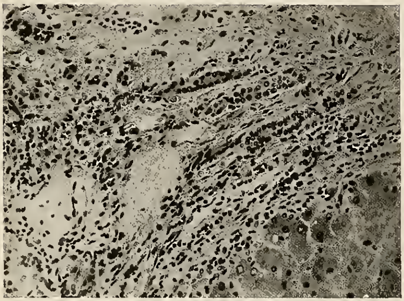
Aorta.—The aorta is normal.

Adrenals.—The cortex is light gray in color and is, compared to the medulla, smaller than usual. The medulla is dark purplish red in color.

Pancreas.—The pancreas is normal.

Stomach.—The stomach is contracted. On opening the stomach it is found to contain a thick tenacious mucus filled with food remains. The wall of the stomach is thickened due to its contracted state. There are a number of small pin-point hemorrhages throughout the mucus membrane.

Intestines.—The intestines are normal except that the Peyers patches in the lower part of the ileum are very prominent and some are congested. The appendix has a lumen to its tip, is filled with feces and is not inflamed. The colon is normal.



Section of Liver

Showing hyperplasia and degeneration of hepatic cells.

Brain.—On removing the calvarium the dura is found to be moderately adherent in the midline. The cerebral meninges are markedly edematous and somewhat congested. From external inspection the brain appears normal. The brain is hardened before section.

Spinal Cord.—There is a large amount of fluid distending the dura surrounding the spinal cord. The spinal cord appears normal on gross section at the cervical enlargement. The spinal cord is hardened without further sectioning.

Microscopical Examination. *Heart.*—Right Ventricle: There is a beginning fragmentation of the heart muscle. Some nuclei are enlarged. The endocardium is thickened. The papillary muscle in the section is half fibrous tissue and there is a beginning fibrosis about the vessels.

Left Ventricle: There is a slight diffuse increase of fibrous tissue. An occasional degenerating muscle fiber is seen which stains a dark purple while some fibers are vacuolated.

Aorta.—The intima is irregularly thickened by a loose broken edematous-looking connective tissue.

Lung.—This presents a striking picture. What at first glance appears to be merely a simple bronchopneumonia in a congested emphysematous lung is seen to be an early stage of giant cell pneumonia. Large groups of alveoli are filled with swollen desquamated epithelial cells. These epithelioid cells are present in large numbers completely filling the air spaces. Many are so fused together that the individual cell outlines cannot be distinguished. Some cells contain two or three nuclei while occasionally a typical multinuclear giant cell is met with. Mitotic figures are both in the desquamated cells and in alveolar cells which are still attached. The cells lining the alveolar walls are swollen and show various stages of desquamation. Their nuclei are either deeply stained or show various degrees of degeneration. The pinkish granular cytoplasm of most of the cells contain varying numbers, generally three to four clear vacuoles. Even some of the still attached alveolar cells show this strange vacuolation. On comparing the typical sections of a giant cell pneumonia (Autopsy No. 8336) similar vacuoles can just be recognized in some of the epithelioid cells. Some alveoli contain red cells and polynuclears are seen in others. Even in the apparently uninvolved areas there are a few epithelioid cells in the air spaces. The bronchi, whose walls are not thickened, contain large quantities of polynuclears. Mixed with these are red cells and a few vacuolated epithelioid cells. The bronchial epithelium is intact.

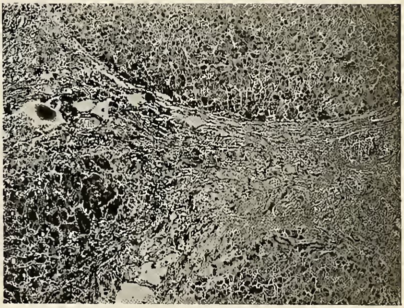
A section of the lower lobe presents a most confusing picture. Almost all the alveoli are filled with some form of edema while some contain epithelioid cells similar to those previously mentioned. In general the edema stains pink, but there are irregular groups of adjacent alveoli which appear bluish. The edema appears as a granular pink material in some alveoli but in others it is a structureless darker homogeneous pink. In places it almost appears hyaline and stains red. In the blue areas there is a dark blue fibrous substance which extends through the pink edema. These firm coagulated masses extend from one alveolus to another and in places irregular projections are seen which extend through the alveolar walls. Some of these form typical fans. The blue strands also extend into the alveoli filled by the granular edema. In some alveoli red cells and polynuclears are present in large numbers, but there is no relation between the kind of coagulum and the number of polynuclears present. The bronchi contain polynuclears and some edema.

Thymus.—Hassall's corpuscles are numerous and large. Some are large and consist of a structureless faint pink-stained granular material surrounded by delicate connective tissue cells. The lymphoid tissue is normal.

Spleen.—The capsule and trabeculae are thickened. Irregular portions of the pulp show intense congestion. The Malpighian corpuscles are numerous and well defined.

Bronchial Lymph Nodes.—Cortex and medulla show irregular areas of intense congestion. The cortical follicles are small, distinct, and well filled with lymphoid cells. In the center of some follicles the lymphoid cells are scanty. In the medulla there are many small cyst cavities lined by a single layer of epithelium. These contain a granular material and in some there are red blood cells.

Mesenteric Lymph Node.—The cortical follicles are prominent and the cortex is filled with mononuclears. In the medulla there are some large cavities lined by a structureless fibrous tissue and filled by a homogeneous faintly pink-staining material. The substance stains blue in a narrow line just inside the capsule. There is a diffuse increase in the connective tissue. In places there are large areas of fibrous tissue in which are many small cyst cavities. The vessels are all congested.



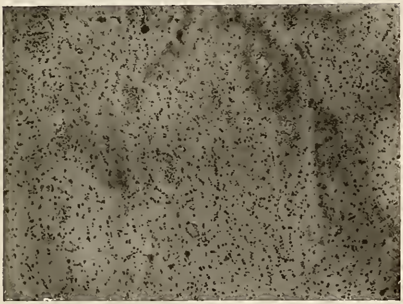
Section through Hepatic Lobule

Pancreas.—The glandular tissue is normal. An occasional island of Langerhans is hemorrhagic, otherwise they are normal. The gland is congested.

Liver.—The portal spaces are filled with varying amounts of edematous connective tissue in which there are small wandering cells, great numbers of regenerating bile ducts and at times many capillaries. New-forming bile ducts are often seen at the periphery of the lobule and also penetrating into the lobes. Between most lobules there are thin strands of connective tissue. An occasional remnant of a lobule is seen entirely surrounded by masses of connective tissue, but as a rule the lobules are not much reduced in size. There is the greatest difference in the appearance of the lobules. In most of them the liver cells show a cloudy swelling, the cytoplasm contains blue granules, the nuclei stain fairly deeply but are not enlarged. In other lobules there is a slight amount of fatty infiltration especially at the periphery. The cytoplasm is granular and swollen, many nuclei have entirely disappeared while, in general, the remaining ones are swollen and stain irregularly. These degenerating lobules are adjacent to those which show only cloudy swelling. There is some regeneration of the liver cells, especially at the periphery of the lobules.

Kidney.—There are a few cortical scars infiltrated by wandering cells. Except for a stray fibrosed glomerulus in a scar the glomeruli are normal. The tubular epithelium is cloudy and swollen. Some tubules in relation to the scarred areas are dilated. Except for congestion, the vessels are normal.

Adrenal.—The reticular zone is absent. There is little fat in the cortex. The chromaffin tissue of the medulla is prominent.



GLIOSIS IN PUTAMEN

Microscopic Appearance of the Central Nervous System.—The brain was cut in macrotomic segments, 1 cm. in thickness in the coronal plane of Marie. The spinal cord was sectioned at the same thickness. The entire brain was cut in serial sections and every fifth section stained by the Pal-Weigert method. Another fifth series was set aside for cellular stain by the iron hemotoxylin method. From one block containing the lenticular nucleus through its middle third, a segment was removed which included the putamen and globus pallidus. This was especially treated by cell stains from which the accompanying photomicrographs were obtained.

Upon macrotomic section the central nervous system, with one exception, namely, the lenticular nucleus on both sides, appeared normal. There were no gross lesions discernible in the cortex or medullary substance. The internal capsule showed nothing demonstrable in the gross. The midbrain, pons and isthmus, cerebellum, and spinal cord were normal. The coverings of the brain showed no pathological condition.

Upon inspection of the macrotomic sections in the gross, those containing the lenticular nucleus showed that this part of the corpus striatum was the seat of some pathological change. The lesions were minute and gave the putamen a spongy appearance. This was more particularly true in the dorsal or superior portions of the putamen and was likewise more pronounced in the region bordering upon the retrolenticular portion of the capsule than in the areas contiguous to the anterior limb of the capsule. Microscopic study of the brain led to the following conclusions:

The cerebral cortex in all areas was normal in its cellular content and in its fasiculation. The vascular status showed no pathological alterations. The corona radiata and the centrum ovale as a whole were normal. The thalamus and hypothalamus showed no changes except that in the latter there was an apparent attenuation in the bundles of fibers which go to make up the ansa lenticularis. The infundibulo-mammillary region was normal; the hypophysis in its glandular and neural portions showed no abnormal changes. The epithalamus, including the habenular region and the epiphysis, were normal. The metathalamus showed no alteration. There was no evidence of degeneration in the fibers of the midbrain and it was difficult to discern any appreciable change in the nucleus ruber. The isthmus, pons, cerebellum, medulla, and spinal cord revealed no pathological alterations in their cell constituency or nerve fibers.

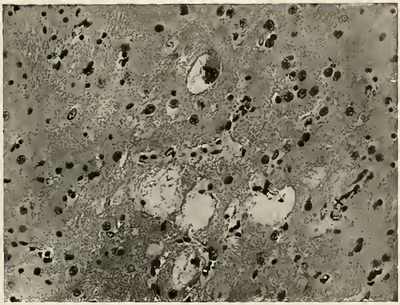
The microscopic appearance of the lenticular nuclei of both sides was striking. Such pathological changes as one noted were confined to the putamen and were more pronounced in the regions of this structure already mentioned in the description of the gross alterations.

A. The Cells in the Putamen.—The larger pyramidal elements, especially, showed a marked degeneration; in some places these cells had been entirely destroyed, leaving in their place a number of lacunae about which were collected ameboid glia cells and other cellular elements which were regarded as plasma cells. In the areas where many of the nerve cells had disappeared in this manner, the putamen gave the impression of a beginning cavitation as illustrated in the accompanying photomicrographs. In many regions the smaller pyramidal cells, as well as those of the larger variety, showed definite tendencies toward degeneration. Their nuclei were eccentrically placed, the cells appeared swollen and their tigroid bodies were difficult to distinguish. From the general picture of the cellular alterations, it seems fair to conclude that a diffuse parenchymatous degeneration was in process of development throughout the entire putamen. In the areas where it had reached its greatest intensity, a process of cavitation had already been established.

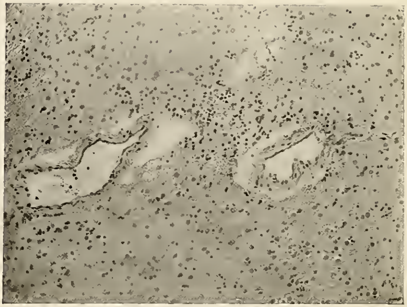
B. The Fibers of the Putamen.—Even in the Pal-Weigert preparations, it was difficult to detect any marked degree of fiber degeneration, although in some places there seemed to be a decrease in the myelin sheaths of some of the axis cylinders.

C. Neuroglia.—Throughout the entire lenticular nucleus, there was a marked degree of gliosis. This reached its greatest intensity, however, in the putamen, the latter standing out in marked contrast to the head of the caudate nucleus, which appeared to present no change in the neuroglia.

D. Blood Vessels and Perivascular Spaces.—Throughout the entire extent of the putamen, the blood vessels seemed to have shrunk away from the adjacent tissue and to be surrounded by large, irregular spaces. These, however, must not be considered as perivascular lymph spaces and, indeed, it is doubtful whether they are of moment in the pathological process at all. They may well be artifacts and are simply mentioned in this connection as their occurrence has been noted in the pathological observations of other reported cases. One reason for making it seem likely that these irregular spaces are artifacts is that their occurrence in this particular brain is not limited to the lenticular nucleus, but rather diffusely scattered throughout several parts of the central axis. The perivascular lymph spaces were not distended nor did they contain any cellular elements which might be considered abnormal, either in amount or character.



Cavitation in Putamen



Vascular Changes in Putamen

The conclusions drawn from the pathological study of this brain are that the lenticular nuclei of both sides were in a state of degeneration which probably was progressive. No portion of the brain showed any unusual biliary or other type of pigmentation.

Summary.—A summary of the cardinal points of this case shows the past history of a boy, fifteen years old, which contained nothing significant, and whose family history was likewise negative. He developed, without discoverable cause, severe nervous symptoms which grew progressively worse until his death seventeen months after the onset. The chief symptoms were a tremor, increased by voluntary movements, hypertonicity of the musculature generally, loss of strength, dysarthria, and toward the end, dysphagia, impairment of bladder and sphincteric rectal control and vasomotor irritability. Physical signs of sensory disturbance were not present. The reflexes were normal. Clearly, this was a case of a motor disturbance with an intact pyramidal system. The patient's condition grew steadily worse and a terminating bronchopneumonia was the final event. The chief lesion at autopsy was an advanced cirrhosis of the liver with slight, though definite changes, in the lenticular nucleus of each side.

Syphilis may be ruled out by the negative history, negative Wassermann, absence of spirochetes in liver, and absence of any other positive evidence.

SYMPTOMATOLOGY OF WILSON'S DISEASE.—Clinically, there are two distinct types of this disease: one *acute* or *subacute*, the other *chronic*. The two types differ very little except in the rapidity with which the invariably fatal termination occurs. The acute cases are apt to have some febrile disturbance; their duration is usually four to twelve months. The chronic cases may last from one to four years after the first appearance of the symptoms.

The striking and characteristic symptoms of the disease are disturbances of motor function.

1. *Tremor* is one of the outstanding features. It is usually one of the earliest and one of the most marked symptoms. It is increased by excitement, or if attention is drawn to it, or by voluntary effort. The range is apt to be fine, at least at the outset, but with volitional movement, the excursions become wider. In the advanced stages, it involves the head and trunk, as well as the limbs. It disappears during sleep.

2. *Spasticity*, or better, hypertonicity, is the second of the cardinal symptoms. Beginning in the limbs, it later spreads to the rest of the voluntary musculature, with one solitary exception—the extrinsic ocular muscle. Their movements remain quick and free. If one takes hold of one of these hypertonic arms or legs and tries to impress on it alternating flexion and extension, he is at once conscious of a considerable degree of resistance in the opposing muscles. It evidently consists in a generalized condition of increased tone in all the muscles indiscriminately.

Every patient reported has shown contracture, usually with the flexion contracture accentuated. This symptom is merely a result of the hypertonicity.

3. *Dysarthria* and *dysphagia* are almost invariably present. They, like the contractures, are the result of the hypertonicity. The dysarthria is characterized by the slurring element without the staccato element of disseminated sclerosis.

4. *Muscular weakness and emaciation* develop, associated with the contractures and immobility of the limbs. A considerable degree of voluntary power, however, has remained in the majority of cases. The weakness and wasting of the musculature are doubtless due in part to the atrophy of disuse and in part to the disturbance of metabolism.

5. The symptoms so far enumerated have all dealt with motor disturbances. Sensory symptoms, except for pains in the limbs mentioned in some cases, have not been observed. Examinations failed to reveal any change in sensibility, nor has any definite alteration of the tendon or cutaneous reflexes been found. Toward the end of most of the cases, however, it has been recorded that the organic reflexes became impaired. This, Wilson attributes partly to mental deterioration, partly to loss of voluntary control over the muscles. His efforts to find a lesion in the cord were quite negative.

6. *Psychical symptoms*, though present in some form in most of the cases, have been very variable. Simple-mindedness, failure of memory and of mental powers, emotionalism, change in disposition, transient delusions, hallucinations of hearing, excitement, narrowing of the mental horizon, facility, docility and childishness are some of the terms used to designate the mental disturbance. Attempts to classify these symptoms have been fruitless, but it seems probable that some psychical disturbance forms an integral part of the clinical picture.

7. *Significant negative signs* are the following: the optic discs and pupillary reactions are normal; there is no nystagmus; there are no cerebellar symptoms.

8. A rather pronounced tendency toward

variability in the symptoms and the resemblance which some of them may present to what is called "functional" disease, led in several cases to a clinical diagnosis of hysteria, the true nature of the disease only becoming clear upon the autopsy table.

ETIOLOGY AND PATHOGENESIS. 1. *Age.*—Progressive lenticular degeneration is a disease of adolescence and youth. The age of the youngest was ten years at the onset; of the oldest twenty-six years. The average age is about fifteen years.

2. *Sex.*—Males and females are about equally affected. It is worthy of note that in the reported cases, menstruation, if established prior to the onset of the disease, almost invariably ceased. The significance of this is obscure.

3. *Heredity.*—Records of family nervous disease or neuropathic tendencies are not found in the reported cases. About three-fourths of the cases are familial. There are two reports of three cases occurring in children of the same family.

4. No constant so-called *predisposing causes* are evident in the histories.

It has been suggested that the disease is an expression of a degenerative tendency—an abiotrophic defect—but it is quite striking that the observers of the reported cases almost uniformly have stated that their patients had normal mental and physical development prior to the onset. It seems highly improbable that a congenital tendency to degeneracy should lie so completely latent for twelve, fifteen, or twenty years.

Speculation as to the intimate nature and pathogenesis of the disease is perhaps interesting, but in the light of the available facts and absence of experimental proofs, rather fruitless. There are one or two philosophical considerations, however, which are worthy of note. There seems to be no analogy for such a distinct morbid entity whose most striking characteristic is a specific association between disease of one of the viscera and disease of a particular part of the gray matter of the central nervous system.

In any discussion of the etiology, syphilis must be considered. Most of the cases recorded occurred in the pre-Wassermann days, but the observers were on the alert to discover signs of syphilis and the absence of positive evidence of either acquired or inherited syphilis is striking. In the more recently reported cases, unquestionably conforming to the syndrome, the Wassermann has been negative. Homén made specially exhaustive efforts to prove that his three cases were syphilitic, but despite his conclusion that they were probably of the nature of a so-called parasyphilitic condition, he specifically states that positive evidence of syphilis, either in parents or children, was wanting. Moreover, the cirrhosis differs from that of hepatic syphilitic cirrhosis in children. The latter is quite constantly associated with ascites.

Alcohol could not be shown to have played a part in the etiology. The presumption, therefore, arrived at by Wilson is that the disease, although familial, is acquired and is toxic in origin, and that the toxin has a selective action on the lenticular nucleus. A very significant analogy to such a hypothesis is furnished by the observations of Werneke, Schmore, Esch and Pfannenstiel who have described cases of icterus gravis or neonatorum. This disease is familial, occurs in the earliest days of life and is fatal in a very short time, the infants rarely living more than a week or two. The interesting feature is that in addition to the general bile-staining of the tissues of the body, there is a selective action in the cerebrum and central nervous system. The lenticular nucleus, the corpus Luysi, the cornu ammonis, the dentate nucleus and the olives, the sensory nuclei of the medulla and pons are specially singled out and stained a bright yellow. The corpus Luysi and lenticular nucleus stain with the greatest intensity. Microscopically, the bile pigment is found in the bodies of the nerve cells. The patients, however, show no symptoms referable to the nuclei. In ordinary cases of jaundice in older people from whatever cause, and in jaundice of congenital bile duct obliteration, no such selective staining of the gray matter has ever been observed as taking place.

There is very little evidence even to suggest that the toxin is of bacterial origin. Wilson examined two of his cases with this in mind, but nothing of pathogenic significance was found.

Speculation as to a possible connection with a disorder of the glands of internal secretion leads nowhere. Whenever examined postmortem, these organs have shown nothing abnormal, except in one case, where the thyroid was described as having a diminished quantity of colloid and some proliferation of the epithelium lining the vesicles.

PATHOLOGY.—For what is known of the gross and microscopic changes in the lenticular nucleus we are largely indebted to Wilson. Using modern technical methods he made exhaustive studies of three of his cases. In several of the cases reported by other observers, examination of the central nervous system proved negative, but these cases belonged to the acute or subacute group and were not subjected to careful study. The pathological changes in the nucleus have regularly been found to be more advanced in the chronic cases. There is, however, a striking absence of parallelism between the intensity of the symptoms and the extent of the nuclear lesion. Accordingly, it must be assumed that the function of the lenticular nucleus may be dynamiically disturbed without much recognizable structural change.

In all the cases with positive findings in the central nervous system, the appearances are so similar that they afford a striking proof of the selective action of some morbid agent. Each of these cases showed a bilateral symmetrical softening of the lenticular nucleus varying from slight discoloration and sponginess of the nucleus through shrinkage and atrophy to complete disintegration and excavation of the ganglion. Sometimes the caudate has been described as a little shrunken but never disintegrated. In five cases the optic thalamus, the internal and external capsules, the claustrum and island of Reil are normal. Homén described patches of thickening in the blood vessels, but Wilson was unable to confirm this. The cerebral cortex, the pons, medulla, and cord have shown no definite alterations.

The microscopic changes consist of an overgrowth of glia cells which afterward break down; there is often an immense increase in glial nuclei; the nerve fibers and nerve cells of the normal nucleus disappear. Phagocytic wandering cells are frequently present. Even when the cavity formation is extreme, there are no signs of an obliterative endarteritis. On the contrary, the blood vessels sometimes appear thinned. Around the vessels gaping spaces make their appearance, possibly from shrinkage of the nervous tissue. Hyaline degeneration in the walls of the blood vessels has also been described. The pons, medulla, cord, nerves, and muscles have uniformly been found negative. Degenerations secondary to the lesion in the nucleus have been found in the ansa lenticularis which is the chief tract connection of the nucleus.

The liver is always cirrhosed. This is, indeed, a *sine qua non* of the syndrome, and a diagnosis of progressive lenticular degeneration should not be made without it. The cirrhosis is always advanced and strikingly apparent. Usually the liver is smaller than normal, although not constantly so. It is firm, hard, preserves its shape and presents the appearance of rounded nodules of liver tissue, clustered together, and separated by cirrhotic bands. The organ is never bile-stained, but is often rather lighter in color than usual. Microscopically, one finds bands of cellular connective tissue extruding out from the portal spaces, sometimes including one, sometimes several lobules, and occasionally there are evidences of an intralobular cirrhosis. Actively regenerating areas of liver cells and so-called regenerating bile ducts are abundant. There is apt to be a moderate amount of fat in the cells.

The question, of course, comes up as to the relation of this form of cirrhosis occurring in young people to the other types of hepatic cirrhosis in children. There are reports of a considerable number of cases of cirrhosis in children in the literature. Howard collected sixty-three cases of cirrhosis of the liver in children. Schlichthorst observed five cases himself and found in the literature reports of over a hundred other cases. The conclusions of these two observers may be summarized as follows:

Cirrhosis of the liver in young people is usually found between the eighth and eighteenth year. Alcohol accounts for about 20 per cent. of the cases, syphilis about 10 per cent., tuberculosis about 10 per cent., and chronic passive congestion, acute infectious diseases (such as scarlet fever, diphtheria, and measles) and malaria about 10 per cent. In the remaining half of the cases no etiological factor can be determined. Atrophic nodular livers were slightly more numerous than large, smooth, fatty, cirrhotic livers. Ascites or jaundice, or both, are common in both forms. Gastric hemorrhages, and evidences of dilatation of collateral circulation are not infrequent. Pyrexia is common. In short, the symptomatology is essentially the same as in adults. It is most apt to be confused with tuberculous peritonitis. There is one very striking difference between these cases of cirrhosis of the liver in children and the cirrhosis of progressive lenticular degeneration, namely, the almost total absence of symptoms in the latter. In only one of the cases of Wilson's disease were there symptoms pointing to the liver. That was one of Wilson's own cases which four years before the onset of nervous symptoms had a transient attack of jaundice, very possibly the ordinary catarrhal jaundice.

A CASE OF FOOD-POISONING OF THE TYPE KNOWN AS BOTULISM*

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Patient: Dr. H. E.; age 36 years; single; dentist.

Family History.—Negative.

Past History.—The patient had the usual diseases of childhood. He denies gonorrhea or syphilitic infection; and has used a very slight amount of alcohol. He uses a moderate amount of tobacco and very little coffee or tea. Four years ago he had an appendectomy; otherwise his health has been perfectly good at all times.

Present History.—On the evening of April 2, 1918, the patient went with a friend to a restaurant and had a dinner which consisted of soup, chicken, string beans, rhubarb, apple pie, and coffee. The next night at ten (about thirty hours afterward) he went to bed because he felt nauseated, although he did not vomit. He awoke the next morning feeling somewhat dizzy and weak, with a feeling of general discomfort. He had breakfast, which consisted of coffee and crackers, and went to his office still feeling dizzy. At ten-thirty that morning (forty-two and a half hours after ingestion of the dinner) he had an attack of vomiting. The vomiting was profuse and not very distressing, but on account of the weakness and dizziness he decided to go home and go to bed. He took a bottle of citrate of magnesia, and three A. B. S. pills, as a result of which he had two slight bowel movements late that afternoon. He continued to be nauseated, weak, and dizzy. The next day his dizziness was more pronounced and one hour after a breakfast of cocoa and crackers he vomited profusely, the vomitus being very sour and with a rancid odor. When seen that afternoon his chief complaint was marked depression and weakness, and a severe vertigo; all objects seemed to be whirling around him. His temperature was normal; his pulse rate was sixty; the abdomen was not distended or tender. The next morning, he awoke feeling much worse; his weakness and dizziness were more profound. In going to the bathroom he noticed that he was unsteady on his legs and would persistently stagger and fall to the left. The following morning, which was about eighty hours after the onset of his illness, his dizziness became much more marked and for the first time he began to see things double; he described the objects he saw in planes, one above the other. His ataxia became much more severe until he was unable to stand or walk without support on both sides. He continued in about this same condition without any change for one week and then began to improve. He first gained in strength; then his dizziness began to leave him; his diplopia gradually became less marked until he saw only distant objects double; he complained of a haziness and confusion of vision, such as one experiences in changing from sunlight to a dark room or the reverse. The ataxia was the last symptom to show improvement. Throughout his illness his mental state was characterized by great clearness and a disposition to observe closely the evolution of his disease; as he expressed it, "All of this without an ache or pain". The patient notices that in the last week (fourth week after onset)he has passed greater quantities of urine than he ever passed before his illness, despite the fact that he is not drinking any more water than usual. He also notices that he has great difficulty in visual accommodation, for when he is looking at a group of objects he must move his eyes slowly from one to the other in order to get the entire picture. His pulse is 84; blood pressure, systolic 118, diastolic 82.

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* Presented at the Clinical Conference of the Neurological Department of the College of Physicians and Surgeons, May 27, 1918.

His companion at dinner was taken sick on Wednesday, April 3, with headache, dizziness, cramps, and diarrhea which continued until Thursday night. Friday she was quite well again. With the exception of the string beans, she ate the same meal as the patient; the patient having eaten both portions of beans.

Physical Examination. Fourth Day of Illness.—The patient was a fairly well-nourished man. Heart and lungs were normal. Blood pressure was systolic, 90; diastolic, 60—giving a pulse pressure of 30. The fundi showed a slight haziness of nasal margin of the right disc; the vessels were not congested. The pupils were slightly irregular in outline, equal, normal in size and reacted promptly to light and accommodation. Eye movements were disturbed by a horizontal nystagmoid movement in both directions and a rotary nystagmoid movement on looking upward. The oscillations to the left were much more rapid and the excursion much smaller than the movements on looking toward the right. The external rectus on the left side seemed to be weaker than on the right side. There was a slight drooping of the corner of the mouth on the right side, although facial movements were performed with ease, and equally well on both sides. The hearing on the left side seemed to be diminished to about half, as compared with the right side. Tuning fork was localized to the right. Bone conduction seemed diminished on the left side. There was no difficulty in swallowing. The visual fields to rough tests did not seem to be contracted and there was no evidence of scotomata. There was no tremor or ataxia of the upper extremities. The reflexes at the wrist and elbow were slightly exaggerated but equally active on both sides. There was no adiadochokinesis. There was persistent spontaneous pass pointing of about three inches to the left with both arms. The abdominal and cremasteric reflexes were present and fairly active on both sides. There was no hypotonia, atrophy, or increase in tone of the muscles of the lower extremities. Normal plantar reflexes were elicited and there was no Babinski or clonus on either side.

Tests for coördination in a recumbent position were well performed without any ataxia. His station showed a very marked Romberg and a tendency to fall to the left. He required a broad base to keep erect. His gait was disturbed by a marked ataxia which was increased on closing his eyes and a tendency to fall to both sides, but especially toward the left.

Sensations for touch, pain, thermal, vibratory, muscle, and joint sensibility were normal.

The urine examination showed a specific gravity of 1020, no sugar, a trace of albumen; no casts, but numerous pus cells; no excess of indican, no diacetic acid or acetone.

Blood Wassermann was negative.

Cerebrospinal fluid showed no cells; globulin content was normal; Wassermann negative. Pressure was not increased.

First Week of Illness.—Ophthalmological report by Dr. Cohen.

Vision: Right eye 20–30 corrected 20–20. Left eye 20–20. Pupillary reactions normal. Field of vision for white and colors normal; no scotomata present. There was marked diplopia. Red-glass test showed a vertical separation of images in depression. Separation increased in adduction. The conclusion from the red-glass test indicates a paresis of the right superior oblique muscle. Nystagmus was present in marked abduction and adduction. It was also present in the normal plane. This nystagmus was of a jerky variety and probably due to fatigue of the muscles. Fundi normal. The paresis of the right superior oblique muscle is probably of a nuclear origin due either to a localized inflammation or hemorrhage, the etiology of which is either luetic or toxic in origin.

Third Week of Illness.—There seemed to be some diminution in the power and bulk of the left masseter muscle.

Fourth Week of Illness.—Otological tests done by Dr. I. Friesner showed the following responses:

Weber is referred to the head. Rinne, both sides positive. Schwabach, slightly shortened both sides. The upper and lower tone limits are normal both sides. Tested with the noise apparatus, both ears hear low conversation readily. With the exception of the slightly shortened Schwabach the hearing of both ears is normal.

Labyrinth.—Station is good, very little swaying. There is no spontaneous pass pointing in the right arm; the left arm pass points two inches to the left. This persists on repeated tests. There is a spontaneous nystagmus of the rotary type directed to both sides, slightly more marked to the right. There is also a vertical nystagmus upward. Rotation to the right is followed by a horizontal nystagmus of fair amplitude, lasting twelve seconds. Rotation to the left is followed by a nystagmus of very good amplitude, lasting twenty-five seconds.

Tests for vertigo: Rotation to the right; the right arm touches, the left arm pass points one inch to the left (spontaneous pass pointing). Rotation to the left; both arms pass point two inches to the left. Caloric reaction of right vertical canals positive but faint, one minute and fifteen seconds. No pass pointing in either arm. Stimulation causes diplopia. Caloric reaction of the right external (horizontal) slightly more marked than that of the vertical canals. No pass pointing in either arm. Caloric stimulation of the right external canal arouses an oblique nystagmus upward. Caloric reaction of left vertical canals positive faint fifty-seven seconds, reaction of external canal more marked. No pass pointing in either arm.

SUMMARY AND CONCLUSION

The hearing is normal. There is a spontaneous nystagmus to both sides and also upward. Functional tests show an interference with all reactions following stimulation of the left labyrinth. Caloric stimulation of the right vertical canals causes diplopia, of the right external canal an oblique nystagmus upwards. The nystagmus following stimulation of the right labyrinth is nearly normal while the vertigo is markedly suppressed. These facts indicate that the lesion is not peripheral but is situated in the brain stem.

Botulism has long been recognized as a distinct type of meat poisoning, and many epidemics have been recorded in the literature, especially in Germany. Spoiled sausage has been regarded as the most frequent cause.

Van Ermengem,[1] investigating an epidemic that occurred in Ellezelles in 1895, established the fact that botulism is due to the action of a specific toxin that is produced by the growth of a strictly anaerobic, spore-bearing bacillus, in a medium which contains animal protein. Experiments by Van Ermengem and others have shown that the toxin was produced only under strictly anaerobic conditions, and that it was rarely produced unless the medium was alkaline in reaction. They found that it was rapidly destroyed in the presence of light or air, or by heating to 80° C. They noted that the presence of the organism was indicated by a rancid odor like that of butyric acid.

In 1904, Landmann[2] investigated an epidemic in Darmstadt, of twenty-one cases that occurred after eating bean salad. He was able to isolate the organism from the beans. He, however, concluded that the beans must have been cooked with pieces of meat.

In 1913, an epidemic of twelve cases occurred at Stanford University, following the ingestion of bean salad that had been prepared from home-canned beans. This epidemic was studied by R. L. Wilbur and W. Ophüls.[3] In this group of cases there was a wide variation in the severity of the disease; some of the patients having only transient disturbances of vision and of swallowing, others such complaints as "jaws being tired," "apparent inability to chew food," "sleepy all the time," and so forth. The gradual onset of the symptoms and their marked variability were the most striking features. They concluded that their cases were due to infection, resulting from eating domestically prepared canned string beans, served without preliminary heating.

Peck[4] reports an epidemic of twelve cases of ptomain poisoning, which he attributed to the ingestion of preserved pears. His clinical description of the cases resembles the clinical picture of botulism so much that there is the greatest probability of their having been due to the toxin of Bacillus botulinus. Eleven of his twelve cases proved fatal and the only common article of food taken was the pears.

Bine[5] reports three cases of botulism due to the ingestion of clam broth. Two of these three cases proved fatal. No pathological studies of the central nervous system were made in his cases.

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[1] *Ztschr. f. Hyg. u. Infectionskrankh.*, 1897.

[2] *Hyg. Rundschau*, 1904, XIV, 449.

[3] *Arch. Int. Med.*, October, 1914.

[4] *South. Calif. Pract.*, vol. XXV, 1910.

[5] *Boston M. & S. J.*, October 18, 1917.

From experimental study, E. C. Dickson[6] concluded that (1) "The presence of animal proteid is not essential for the production of the toxin of botulism; (2) the toxin may be produced in a medium made from string beans or peas; (3) an acid reaction of as much as 3.2 per cent. to phenolphthalein does not prevent the formation of the toxin." His conclusions in regard to the pathology were also of interest. He showed that "the toxin produces some disturbance of the circulatory system which leads to hyperemia and hemorrhage in the meninges and in the central nervous system, and to thrombosis in the arteries and veins of the meninges and the central nervous system; that the lesions in the nerve cells are not due to a specific action of the toxin on the cell, but are secondary to disturbances of blood supply."

In the one fatal case studied by Wilbur and Ophüls, they found a wide-spread thrombosis in the blood vessels at the base of the brain and in some of the blood vessels in the substance of the brain. Thrombi were also found in the substance of the brain stem, some of them in close proximity to the important nuclei." In the cortex and the cerebrum there was much hyperemia but no thrombi. Van Ermengem made a careful study of the tissue of the animals of his original experiment and he described as the most important lesion a type of degeneration of the Nissl granules in the ganglion cells. This degeneration was not present in the case-studies by Wilbur and Ophüls.

Van Ermengem has summarized the symptom-complex presented in botulism as a neuroparalytic symptom-complex with disturbances of secretion and with symmetrical partial or complete motor-paralyses which apparently have their seat in lesions of the central nervous system. The first symptoms usually do not occur earlier than twelve to twenty-four hours after the ingestion of infected food, although they may appear much later. In contrast to usual types of ptomain poisoning there is apt to be little evidence of acute gastro-intestinal disturbances; constipation is more frequent than diarrhea.

There is usually a decreased, but there may be an increased, secretion of saliva and mucus in the mouth and in the throat; when mucus is present it is extremely viscid. There is early disturbance of the external and internal muscles in the eyes, which manifests itself by ptosis, mydriasis, disturbance of accommodation, diplopia, and strabismus. General muscular weakness is common. There may be dysphasia or aphonia and there is usually difficulty in swallowing. The absence of sensory disturbances is characteristic.

When death occurs it is usually from respiratory or cardiac failure. When recovery takes place there is a long and tedious convalescence.

The condition closely resembles gelsemium and hyoscin poisoning, polioencephalitis, cerebral syphilis, and bulbar palsy. A number of cases, particularly if all have partaken of a common article of food, should arouse one's suspicion.

An isolated case of great interest in connection with my case, on account of its similarity, is reported by Percy G. Styles,[7] who himself was affected. He had eaten lunch, consisting of minced chicken on toast, mashed potato and coffee, and jelly with whipped cream. Within about forty-five minutes he became conscious of a growing inertia. After a momentary sensation of flushing, one hour later, he reacted to chilliness and began to sway on his feet. He next noticed a peculiar hyperesthia of the pharynx. On account of faintness he was compelled to lie down. An attempt to sit up after an interval of rest caused streams of cold sweat and a feeling of utter helplessness. After about one and a half hours a sudden onset of nausea and scant vomiting occurred. Half an hour later he again vomited profusely. A third attack of vomiting occurred about one and a half hours later, and he developed a marked vertigo and nystagmus. The next morning he developed intermittent heart action. His vertigo and nystagmus continued unchanged. He again vomited in the afternoon of that day and later passed the first urine in more than twenty-four hours. In the next few days he gained strength, and the vertigo, though still severe, was diminished. Four days after the onset of his illness on attempting to walk he found that an ataxia existed to such an extent that he could scarcely keep his feet. The ataxia was accompanied by dizziness and confusion of vision. On the eighth day of his illness he developed difficulty in speech and swallowing, and quantities of viscid mucus were secreted in his mouth. The nystagmus became limited to momentary onsets, and was replaced by a peculiar diplopia, the images being displaced and tilted about fifteen degrees from parallel. Convalescence was tedious. After about ten weeks, normal walking was attained through diligent effort.

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[6] *J. Am. M. Ass.*, August 7, 1915.

[7] *J. Am. M. Ass.*, December 27, 1913.

The unusual feature in this case is the extreme rapidity with which the toxin manifested itself. In this regard we must consider individual susceptibility as well as the degree of concentration of the toxin and the rapidity with which it is absorbed.

Seven weeks after the onset, my patient still presents some difficulty in accommodation; ataxia in finer coördinate movements; slight spontaneous pass pointing to the left with the left arm; nystagmus on fixation in both lateral planes and also on upward fixation; and atrophy of the left masseter muscle.

From a study of the literature there seems to be little doubt that the Bacillus botulinus can grow and produce its toxin in the absence of animal protein material. The onset, course, symptoms, and protracted convalescence in my case would suggest poisoning with the toxin of the Bacillus botulinus, although only an isolated case in which no attempt was made to recover the organism from the infecting material.

In view of the fact that my patient ate both portions of the string beans and that only he showed the typical symptoms while his friend merely had a gastro-enteritis, it seems highly probable that the string beans contained the infecting material.

DISCUSSION

Dr. S. Philip Goodhart.—The very complete and lucid presentation of this case leaves little for discussion. Personally, I have never seen a case of botulism and, although I have read accounts of the epidemics, I doubt that I would have been able to make the diagnosis as botulism in this isolated case. I had supposed that the Bacillus botulinus was found only in animal protein, and here we have clearly a case due to vegetable protein. I believe, however, that there are probably many more cases than are recognized as such. Here is a man who apparently ate infected vegetable protein served at a restaurant widely patronized. Are we to assume that he was the only person infected? Does the organism attack only susceptible persons? I have no doubt that there are many isolated instances of this disease, but that the symptoms—the incoördination, the nystagmus, and cranial nerve palsies—are mild and transitory. I am convinced that Dr. Grossman's case is one of genuine botulism and I think we should all be on the lookout for this type of case. I might add that the symptom-complex would be most difficult to reconcile with any other than a lesion of toxic nature. Similar symptoms and a somewhat analogous pathologic picture in fatal cases have been observed in malarial infections. Here again the analogy—infecting organism, toxin, and consequent vascular changes—is evident.

Dr. Morris Grossman.—We tried to ascertain if any more cases of illness of this sort had been reported to the restaurant proprietor, but naturally he denied other instances.

Although numerous epidemics have been reported, this is the second isolated case in the American literature. In England, at the present time, they are having many isolated cases. In April of this year, Harris reported in the *Lancet* seven isolated cases all derived from canned material of various kinds. In that same issue, Hall presented ten cases as toxic ophthalmoplegia; in a note at the end of his communication he stated that these were probably cases of botulism. It would seem to me, as Dr. Goodhart suggested, and in view of the many cases that are being encountered abroad, which are undoubtedly due to the economic conditions in the canning industry, that we must be careful not to overlook possible cases of infection derived from this source, as well as those that may occur on account of changed personnel in the canning industries in this country.

THE SUPRA-OPTIC CANAL IN ITS RELATION TO AN INTERPRETATION OF CHOKED DISC

By FREDERICK TILNEY, M.D.
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None of the explanations concerning the phenomena of choked disc is wholly satisfactory, and indeed no single explanation may be entirely without its defects. Several theories have been advanced, of which the most prominent are the toxic theory of Leber and the Manz-Schmidt-Rimpler mechanical theory. According to Leber's view diseases of the brain, as well as diseases in other parts of the organism, are capable of producing toxins which, after gaining access to the cerebrospinal fluid, make their way into the portion of the subarachnoid space which invests the optic nerve. In this position the toxic fluid acts upon the nerve fibers as an irritant and thus produces inflammation, with the end result of an optic neuritis in and about the nerve head and final edema of the disc.

In contrast to this conception, which depends upon a more or less theoretical toxicity of the spinal fluid, the Manz-Schmidt-Rimpler theory seems to be on firmer ground, since its conclusions are based upon a mechanical interpretation of the anatomical facts. This view concerning the causation of choked disc recognizes the following conditions: first, a continuance forward upon each optic nerve of the subarachnoid space, which is known as the intervaginal space; second, this portion of the subarachnoid space is in direct connection with the subarachnoid space investing the brain; and third, the cerebrospinal fluid may pass from the larger spaces about the brain into the smaller extracranial spaces surrounding the optic nerves.

The mechanical theory, making use of these anatomical facts, maintains that any pathological conditions which tend to increase the pressure upon the cerebrospinal fluid within the skull would, of necessity, force more of this fluid into the intervaginal space surrounding the optic nerves than is normally present. The new pressure upon the optic nerve, determined in this way, constricts the nerv, impedes the drainage in the hemal and nonhemal channels, compresses the nerve fibers as they leave the eyeball through the cribriform plate and so produce a papilledema. Following this conception to its logical conclusion, any mass or body within the cranial cavity not normally belonging there should produce a choked disc. This explanation undoubtedly offers a most convincing explanation of papilledema. On the other hand, it has not taken into account all of the anatomical possibilities, for it is only recently that the knowledge of another factor, which must be given its proper value, has been acquired.

Several years ago the writer presented a paper to the American Association of Anatomists in which he described the presence of a small canal extending out from the cephalic extremity of the third ventricle along the chiasm and the optic nerve. This I called the *supra-optic canal*, and as it was first observed in studying the forebrain of the domestic cat the detailed description which I give of it in this animal will serve as a paradigm of all the other forms studied. In the cat the canal projects laterad from the cephalic extremity of the ventricle across the optic chiasm and for a considerable distance along the optic nerve on either side. It is a distinct continuation of the third ventricle. It is lined with ependyma and measures about one centimeter in length. A small probe may be passed into it for this distance. It is situated upon the dorsum of the chiasm and in this position extends along the optic nerve. The canal is marked upon the surface by a ridge running along the dorsum of the chiasm and the optic nerve; this is the *supra-optic ridge*. I have observed this canal in the following forms: Squalus acanthus, Mustelus leavis, Lepidosteus osseus, Rana pipiens, Menobranchus, Iguana tuberculata, Gallus gallus, Botaurus lentiginosus, Didelphys

quica, Bradypus tridactylus, Dipus ægyticus, Dasyprocta agouti, Mus decumanus, Lepus sylvaticus, Sphingurus prehensilis, Mephitis mephitica, Odocoelus hemionus, Odocoelus virginianus, Oryx beatrix, Ovis tragelaphus, Ovis aries, Castor canadensis, Mirounga (Macrorhinus angustirostris), Nasua narica, Ursus horribilis, Canis latrans, Canis familiaris, Genetta vulgaris, Felis domesticus, Felis pardus, Felis leo, Lemur macaco, Macacus cynomolgus, Nyctipithecus trivirgatus, Babuin cynocephalus, Hylobates hoolock, Simia satyrus and Homo.

From the fact that this canal is phyletically so constant one would expect some definite ontogenetic explanation of it. This was sought in the embryology of several forms and found to be thoroughly consistent in all of them. In giving a description of the development of this canal it will be necessary to go back to the early stage of eight somites in the cat embryo at which time the neural tube is still widely open in the encephalic portion of the tube and this cephalic extremity has developed a marked bilateral expansion to form the primitive optic vesicles. These vesicles contain a large cavity, the prosencephalic ventricle. A little later the primitive optic vesicles show a constriction which causes the appearance of two small evaginations now recognizable as the definitive optic vesicles. Each of these vesicles contains a recess which communicates with the prosencephalic ventricle. The process by which the primitive optic vesicles develop includes an actual reduction in the size of the original optic evagination and a marked relative increase in the other structures constituting the prosencephalon. With this reduction in size of the optic vesicle two features are discernable, first the relatively rapid reduction of the proximal portion of the two vesicles and second, the tendency of each vesicle to preserve at its distal extremity most of its primitive configuration. In consequence of these changes the two optic vesicles are in relation with the forebrain by means of a constricted, transverse, connecting piece destined later to form the stalk of either eyecup.

From the stage of 21 somites to that of 4.5 mm. (26 somites) the optic stalk shows a progressive attenuation which is conspicuous on the ectal surface, but more prominent internally. The result of this is that the optic evagination is now in communication with the forebrain by means of a constricted canal. From the orifice of this canal into the prosencephalic ventricle a deep groove proceeds ventrocaudad to terminate in the evagination of the infundibular region; this is the optico-infundibular groove. The stalk itself, upon the external surface, shows no other prominent features, with the exception of the fact that dorsally it is well defined and circumscribed by a groove which sets it off from the rest of the brain wall in this region. Ventrally, however, no such line of demarcation can be detected and the ventral surface of the stalk passes over into the floor of the forebrain without perceptible change to indicate the transition.

In the 4 mm. embryo the optic stalk extends obliquely outward from the plane of the floor plate to the ventral surface of the optic cup, forming within the wall of the prosencephalon an angle of about 45°. The optic vesicle at this stage of development is ovoid in shape and as yet shows no tendency to the process of evagination which ultimately results in the formation of a double-layered eyecup. This process of evagination makes its appearance at the stage of 6 mm. at which time an indentation is present on the ventral surface of the optic vesicle near its center. This slight depression may be traced for a short distance proximally along the optic stalk, which latter has undergone still further constriction. A corresponding decrease is observed in the orifice of the optic canal by means of which the forebrain ventricle is in communication with the recess of the optic vesicle.

The developmental conditions in this stage clearly indicate that the cavity of the prosencephalon communicates with the recess of the optic vesicle through a short canal transversing the optic stalk. It is to this canal that attention is especially drawn in the further evolution which results in the adult condition giving rise to the supra-optic canal shown in Fig. 2. A coronal section through the optic vesicle and forebrain of a 4 mm. embryo shows the condition above described in which the short, constricted stalk contains the canal by means of which the prosencephalic ventricle communicates with the recess of the optic vesicle.

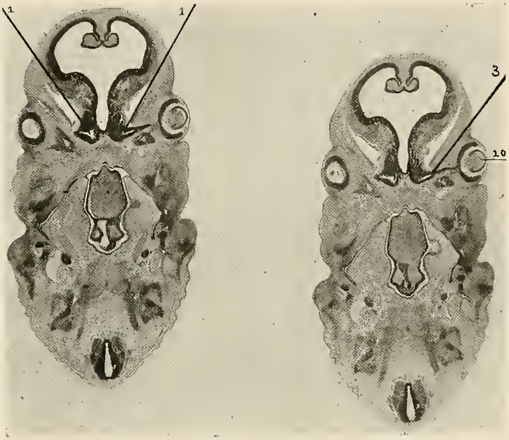

Fig. 1. Section of 10 mm. Cat Embryo

Showing (1) Supra-optic canal (3) Optic nerve (10) Eyecup

In passing to the stage of 10 mm., all the processes which have their inception at the stage of 4 mm. have been carried to their ultimate embryonic state. The optic stalk and vesicle show one change of considerable importance. Along their ventral surface there now appears a broad, shallow groove, which upon passing into the ventral surface of the optic vesicle, takes on the form of a small fossa. The groove in the stalk becomes the chorioid fissure, while the fossa in the vesicle marks the inception of the invagination of the eyecup. The stalk is still without sharp demarcation in its relations to the floor of the diencephalon; it still contains a small canal connecting the cavity of the third ventricle with the recess in the vesicle (Fig. 1).

These conditions remain unchanged until the stage of 14 mm. at which time the optic stalk has become greatly reduced in size in its relation both to the optic vesicle and the interbrain. In form it is a slender peduncle of considerable length extending between the diencephalic floor and mesial ventral surface of the eyecup. At this period the eyecup is completely invaginated, forming the two-layered epithelial structure between the layers of which there is a small fissure-like cavity still maintaining communication with the canal in the optic stalk and thus affecting a continuance of the ventricular system from the third ventricle into the interlaminal fissure of the eyecup. The canal in the optic stalk is much reduced in size and appears about to give up its direct communication, particularly near the middle of the optic stalk. No optic fibers as yet are seen in the stalk and it is not until the subsequent stages of development that the optic axones make their appearance along the ventral surface of the optic stalk. When they begin to grow inward their collected bundles give rise to a marked increase in the size of this portion of the stalk while the dorsal area in its entire extent changes but little from its primitive condition. After all of the optic fibers have entered into the stalk they occupy not merely its ventral portion but also pass into a more dorsal position, thus reducing the dorsal extent of the optic stalk and forcing the remnants of the canal into its ultimate dorsal position.

In the stage of 26 mm. in the cat the ingrowth of the optic fibers is pronounced. It has caused a further decrease in the size of the canal and the stalk, particularly in the middle portion of the latter. Indeed, so much has the canal become reduced in size that now in this portion of the stalk mere remnants of its walls may be discerned while the canal itself is practically obliterated. The primitive optic canal becomes divided into two portions: first, a peripheral portion extending out from the stalk near its proximal extremity and communicating with the much-reduced interlaminal fissure of the optic cup. The second portion is in a proximal position and extends from the diencephalon outward along the dorsal surface of the stalk for about one-quarter of its entire length; this is the *supra-optic canal.* It maintains relatively the same relation throughout all the later stages of development, manifesting no changes other than an attenuation both in its length and transverse diameters. It always remains in a position which lies dorsal to the chiasm and in its peripheral continuation is dorsal to the optic nerve fibers. Its floor is formed by the bundles of the optic nerve and optic chiasm, which fibers also constitute its lateral boundaries while its roof is formed by a thin plate of ependyma to which is applied a layer of the pia mater. Its lining is formed by an ependymal extension of the lining of the third ventricle.

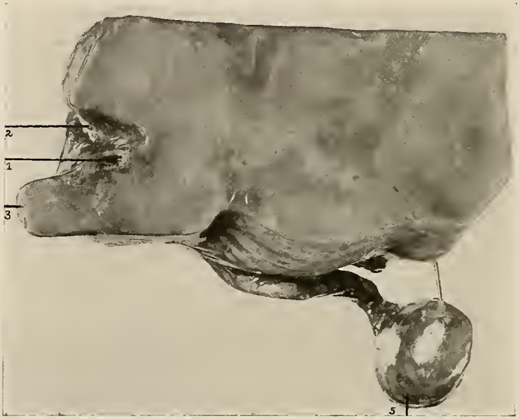

Fig. 2. Coronal Section through Optic Vesical and Forebrain of 4 mm. Cat Embryo

Showing (1) Supra-optic canal (2) Supra-optic crest (3) Optic nerve (5) Infundibular process

It is interesting to note in connection with the ontogenetic history of this canal that there are certain forms in which the eye is rudimentary and in which the optic vesicle does not proceed to its ultimate stages of development. In such animals the connection between the third ventricle and the recess of the eyecup is still maintained by a small canal which passes out upon the optic nerve. This is the case in Necturus in which, as has been shown by Herrick, Osborne and Kingsbury, the rudimentary condition of the eye is accompanied by a similar condition of the optic nerve, the latter retaining the primitive lumen of the optic stalk. Thus, the phylogenetic and ontogenetic history of the supra-optic canal make it sufficiently clear that this extension of the third ventricle may be considered a well-defined entity in vertebrate morphology.

The anatomical conditions observed in the adult cat are best illustrated by a reconstruction of the ventral portion of the lateral wall of the third ventricle in Fig. 2. At the cephalic extremity is situated the optic nerve (3). Across the dorsal surface of the nerve runs a transverse ridge which may be traced from the extreme lateral portion of the reconstruction to the sagittal plane; this is the supra-optic crest (2). Below this is the opening upon the optic nerve of the supra-optic canal (1), so that under normal conditions the position of the supra-optic canal is marked upon the surface of the chiasm and of the optic nerve by this transverse crest. The structures lying caudal and ventral to the canal in this reconstruction are, in the order mentioned, the optic nerve (3) and chiasm, the postchiasmatic eminence, the infundibular stalk, the infundibular process (5), the postinfundibular eminence, and the mammillary body, while in a lateral position is seen the protuberance of the lateral eminence.

A cast of the third ventricle in the domestic cat, made from the same specimen as the reconstruction already shown, illustrates more clearly the relations and extent of the supra-optic canal. This reconstruction is shown in Fig. 3 and presents a ventral view of the ventricle cast.

Its most cephalic structure indicates the position of the pre-optic recess. Laterad this recess is traced into two long projecting cornua representing the supra-optic canal. Caudal to the supra-optic recess is the portion of the ventricle lying above the chiasm, following which is the postchiasmatic recess (6) with the recess of the infundibular process (8). The supra-optic canal in Felis leo is shown in Fig. 4 and in Felis domesticus in Fig. 5.

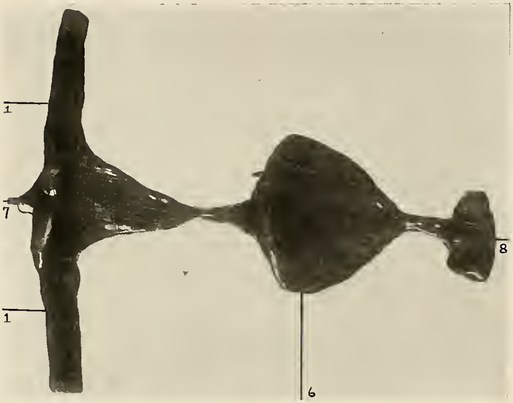

Fig. 3. Cast of Third Ventricle of Cat

Showing (1) Supra-optic canal (6) Postchiasmatic recess (7) Pre-optic recess (8) Infundibular recess

The supra-optic canal in the human presents essentially all of the relations cited as characteristic of it in the cat. The rotation ventrocephalad of the hypophysis causes a slight alteration in the relations of the several recesses of the third ventricle. This, however, does not affect the general disposition of the two supra-optic canals or the pre-optic recess. The canal extends laterad over the chiasm and optic nerve from the cephalic extremity of the third ventricle for a distance varying in different subjects. In some instances the length of the canal from this orifice into the ventricle is .5 cm.; in other instances it may be as long as 1 or 2 cm. A probe introduced into the preoptic recess can, with gentle pressure, be passed into this canal. It is much more extensive in the infant and child than in the adult, but is present at all periods of life. It is usually symmetrical on both sides, being of about equal length on the right and left. Its lining is formed by the ependymal extension of the third ventricle and it is marked upon the surface by a slight elevation extending from the midsagittal plane laterad along the chiasm and then along the nerve to the termination of the canal; this is the supra-optic crest. Often it is difficult to discern this crest in the dissected specimen, particularly when search is made for it by means of deflecting forward the stumps of the optic nerve and the optic chiasm. The pressure resultant upon this deflection causes an obliteration of the supra-optic crest and consequently destroys the contour which is clear upon sagittal section passing through the chiasm and the nerve.

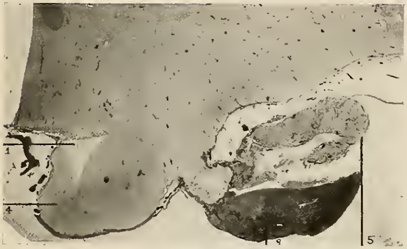

Fig. 4. Section through Brain of Felis Leo

Showing (1) Supra-optic canal (4) Optic chiasm (5) Infundibular process (9) Pituitary gland

A canal occupying such a position in relation to the optic chiasm and optic nerve can, under certain circumstances, undergo such changes, particularly as the result of increase in the amount and pressure of the cerebrospinal fluid contained in this canal, as to compromise the optic fibers passing beneath it. Furthermore, it is not only possible that pressure might be brought to bear upon these fibers direct, but drainage channels making their way through this part of the nervous system would, because of this pressure, undergo changes and be less competent in their drainage capacity. Should this pressure be carried to an extreme degree drainage might be entirely obstructed. That such is the actual case is well illustrated by an instance of internal hydrocephalus in a child. For purposes of control and comparison the supra-optic canal is shown in Fig. 6, a photomicrograph of a sagittal section through the optic nerve in a three-year-old child.

In this figure, the optic nerve (3) appears as a long projection in the dorsum of which near the junction of the nerve with the base of the brain is the supra-optic canal (1). Its surface expression is given by a bulging forward in the form of a fold which in its continuity would represent the supra-optic crest (2). Attention is particularly directed to the marked prominence of this crest in the normal child and also to the relative thickness of its roof. The position which the supra-optic canal bears to the underlying optic fibers obviously lays this latter liable to pressure should that canal become distended due to increase in the amount of cerebrospinal fluid in it.

Fig. 7 is taken from a three-year-old child suffering from internal hydrocephalus. The photomicrograph is made from a sagittal section through the same relative portion of the optic nerve, as Fig. 6, and the magnification in these two illustrations is the same.

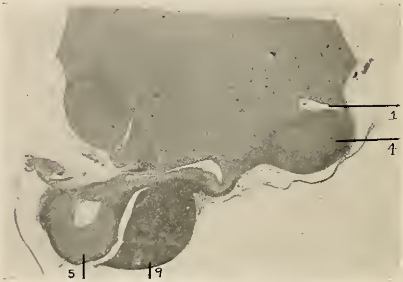

Fig. 5. Section through Brain of Felis Domesticus

Showing (1) Supra-optic canal (4) Optic chiasm (5) Infundibular process (9) Pituitary gland

It is apparent that the supra-optic canal in the case of internal hydrocephalus has undergone marked dilatation; this is evidenced not only by the expansion of the canal, but also by the very significant fact that the supra-optic crest has become entirely obliterated as the result of pressure. This may be directly the result of the increased pressure in the cerebrospinal fluid affecting the supra-optic canal, but it is probably in part also due to the generally increased pressure of the hydrocephalus both in the lateral and third ventricles. The section through the hydrocephalic canal suggests a distinct compression of the optic fibers lying ventrad to the expanded canal and offers a possibility for obstruction in the drainage channels.

CONCLUSION

The expansion of this canal might not be sufficient evidence to denote this dilatation but the condition of the ependyma which is in a state of degeneration and desquamation still further bears this out. The desquamative process is apparent in all portions of the circumference of the canal. Similar dilatation has been observed in all of the cases of internal hydrocephalus which I have been able to study post-mortem; the number, however, is unfortunately small but the uniformity in the occurrence of this dilatation in every case examined is striking. It is also important to note that each one of the cases of internal hydrocephalus showing this distention of the supra-optic canal was accompanied either by a choked disc or optic atrophy with or without blindness. It seems altogether probable, therefore, in the light of mechanical possibilities, that a distention of the supra-optic canal as the result of increased tension upon the cerebrospinal fluid may produce changes which manifest themselves clinically in the alterations of the optic disc, either as primary effects indicated by papilledema or as secondary effects, namely, optic atrophy. The direct cause of the increased tension upon the cerebrospinal fluid would depend upon a generalized increase of this tension in the ventricular system of the brain, either in some part or in the entire system. The pathological condition capable of producing such generalized increased tension is internal hydrocephalus, which condition would therefore be capable of producing marked alterations in the optic discs of the types already mentioned. Internal hydrocephalus is the result of the many disturbances affecting the ventricular system. Thus, it may arise from an excessive secretion of the fluid, from a marked decrease in the absorption of the cerebrospinal fluid, or again it may be due to a blocking of the natural passages of escape for the fluid from one part of the ventricular system to another, as for example, in the aqueduct of Sylvius or in the region of the fourth ventricle.

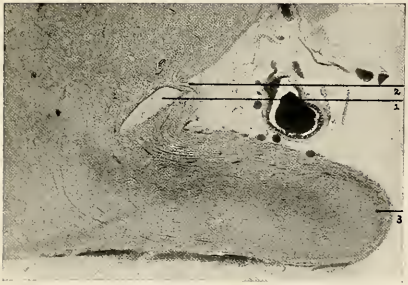

Fig. 6. Sagittal Section through Optic Nerve in Three-year-old Child

Showing (1) Supra-optic canal (2) Supra-optic crest (3) Optic nerve

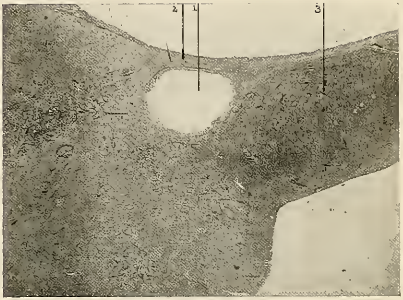

Fig. 7. Sagittal Section through Optic Nerve in Three-year-old Child Suffering from Internal Hydrocephalus

Showing (1) Supra-optic canal (2) Supra-optic crest (3) Optic nerve

Among the more common causes which give rise to a blockage in the ventricular system, tumors of the brain have perhaps received the most attention. By blocking the ventricle the neoplasms may give rise to internal hydrocephalus and, therefore, it may be expected that changes in the optic disc should be manifest in many lesions of this kind. The occurrence of choked disc is variously estimated as present in from 80 to 90 per cent. of all cases of brain tumor, the remaining 10 to 20 per cent. show no such disturbance. Papilledema, or optic atrophy, on the other hand, is much more constantly present in internal hydrocephalus due to any cause. Opinion is now turning toward the belief that it is the complicating internal hydrocephalus occasioned by the tumor which produces changes in the optic disc. Such a view would explain the absence of choked disc in the percentage of brain tumor cases above noted. It is possible that some new growths may exist without compromising the ventricular chambers or blocking the natural avenues of escape of the spinal fluid. This would be particularly true in endothelioma where the surface pressure is often so diffuse as to cause no effect upon the ventricles. Small tumors, on the other hand, if rightly placed, as, for example, in the aqueduct of Sylvius, might readily occlude the passage between the third and fourth ventricles, and thus cause a retention of the fluid in the ventricular system. The causation of choked disc, from this standpoint, would require the establishment of an internal hydrocephalus. This does not in any way preclude the possibility of the production of papilledema due to increased pressure in the intervaginal space. So far as our knowledge goes today, it seems permissible to conclude that choked disc may be the result of the following mechanical disturbances in the anatomical relations:

1. Intracranial pressure upon the fluid in the subarachnoid space causing distention of the intervaginal spaces surrounding the optic nerves, which is the Manz-Schmidt-Rimpler mechanical theory.

2. Internal hydrocephalus may produce a distention of the supra-optic canal and in this way put pressure upon the optic nerve and chiasm, obstructing the drainage channels of these structures.

3. Both of these mechanical disturbances may be simultaneously operative.

The demonstration of the extension of the supra-optic canal furnishes another anatomical element which must be taken into account in any explanation of papilledema on the mechanical basis. In this light, another point of view is given to the significance of choked disc, the interpretation of which depends upon the etiological factors whose cause is an internal hydrocephalus. The theory of Leber, known as the *toxic theory* in the causation of papilledema, is not without the support of many facts to warrant its acceptance, but here the factors causing the disturbance are not mechanical, at least as the theory was originally conceived by its author. It is possible, however, that toxins may act directly upon the ependymal lining of the ventricle in such a way as to cause a swelling in its epithelium and thus, by blocking the avenues of escape of the cerebrospinal fluid produce an internal hydrocephalus. In such an event toxic substances acting directly to produce changes in the ependyma would secondarily give rise to an internal hydrocephalus with its attendant changes in the optic disc.

NEURORLOGICAL BULLETIN

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SPONGIOBLASTOMA WITH UNUSUALLY RAPID GROWTH FOLLOWING DECOMPRESSION*

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Patient: G. W.; female; twenty-nine; single.

The patient was admitted to the Montefiore Hospital on August 4, 1917. She always enjoyed fairly good health except for severe constipation with accompanying headaches. The latter were accentuated during menstrual periods. During her severe headaches she frequently vomited. She had never suffered from any acute infectious disease.

Mental condition supposedly adequate and satisfactory.

Present Illness.—Three weeks before admission to the hospital, while bathing in the surf, she was thrown several times by breakers and on one occasion struck her head. Following this she suffered from some headaches in the frontal region of the head, but this was not considered more unusual than her other headaches. There was no hemorrhage from the nose, ears, or mouth and no disturbances in motor power, speech, or mentality. About five days later, she suddenly developed weakness in the left arm and leg, and this became gradually intensified. There was no fever. The Board of Health was consulted, they diagnosed the condition as one of poliomyelitis, and the patient was sent to Willard Parker Hospital. While there the medical staff apparently came to the conclusion that the original diagnosis was erroneous. Upon the advice of Dr. Frederick Tilney, the patient was released from the Willard Parker Hospital as a non-contagious case. During her stay in the hospital she vomited occasionally (projectile in character). The headaches became more severe and were localized in the right frontal region. Her left-sided paralysis became more marked and was of a spastic type. There were no sphincteric disturbances and her speech and general mentality were good. She showed a tendency to lapse into stupor, from which she could be easily aroused.

Examination on Admission.—Mental state was dull, listless, and somnolent. Her replies to questions were slow and monosyllabic. She displayed very marked desire to be left alone. Her replies to questions were relevant, coherent, and she exhibited no evidence of motor speech defect. She appeared to be quite nauseated but did not vomit. Respiration and temperature were normal, pulse 74. Head could be retained in the middle line, but was thrown to either side. She complained of considerable pain in the back of the neck, and there was slight rigidity of the neck muscles. She was well oriented, and her memory was good. She recognized objects and handled them correctly with her right hand.

Head.—Olfactory sense was intact. Vision was good and equal on both sides, no hemianopsia, no limitation of visual fields. Examination of the eyegrounds revealed definite optic-neuritis of equal intensity on both sides. Eyes—right pupil was somewhat larger than the left, reaction of right not as prompt as left. No ptosis. No extra-ocular palsies or limitation. No corneal anesthesia. Upon showing the teeth or smiling there was definite paresis of the lower left two-thirds of the face. The tongue, however, protruded in the midline and presented no tremor. Audition was good and equal. There was no injury to ear drum or hemorrhage. Speech was slightly lisping, but this is probably a personal characteristic. Mastication and deglutition were unimpaired. Neck muscles presented no differences in motor power.

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* Presented at the Clinical Conference, April 29, 1918

Extremities.—The left upper extremity presented at first a spastic paralysis in flexure position, but this soon began to lose its rigidity and became flaccid. The left lower extremity showed a flaccid paralysis. When asked to make voluntary movements she was able to accomplish very little flexure motion in the distal parts of the lower left extremity. The right upper and lower extremities presented adequate motor power.

Reflexes.—Left upper and lower abdominals were absent. There was definite Babinski on the left foot, supported by Chaddock and other extensor responses of the big toe. Tendon reflexes were increased on both sides, but more so on the left. There was slight exhaustible ankle clonus on the left.

Sensation.—Tactile was absent in the left upper and lower extremities and lower two-thirds of left face. Present on the trunk. Pain (pin-prick) apparently more acute on the left.

Deep Sensibility.—Deep pressure was present but postural sense was lost in the distal parts of the left upper and lower extremities.

Asteriognosis.—Present on the left, even when the fingers were pressed deeply against the object, so that, in spite of retaining deep pressure sensibility, she was unable to make out the size, form, or texture of the object.

Vasomotor and Trophic.—There were no muscular atrophies and no disturbances of circulation. Lumbar puncture—clear fluid removed under moderate pressure. Normal cell count, but moderate increase of globulin. Wassermann was negative.

The diagnosis was that of a neoplasm situated subcortically in the parietal lobe and extending forward into the frontal lobe.

EXAMINATION. Three Days after Admission.—The patient entered a semicomatose state which became more intensified during the day. Breathing was labored and at times assumed a Cheyne-Stokes character. The face was generally flushed, and the head could be moved without any difficulty, there being no rigidity and no pain. The choked discs were more marked on both sides. The hemiplegia became practically complete and more spastic. Blood count—leucocytes 20,000.

In the evening a right cerebral decompression was performed by Dr. A. A. Berg, by the removal of a large bone flap. The bone was greatly thickened. After incision of the dura there was a marked protrusion of the brain tissue. Aspiration in direction of the ventricles was made and a few fragments of brain tissue were obtained for microscopical diagnosis.

Following the operation there was no change in the physical signs other than that the swelling of both optic discs was considerably lessened. The left hemiplegia was complete with diminished epicritic and protopathic sensation on the entire left side of the body. The mental condition was unchanged.

The subsequent course of the disease was a rapidly progressive one. The left upper extremity developed flexure contractures of both elbows, shoulders, wrists, and finger joints. There were likewise contractures of the joints of the lower extremities. There was absolutely no muscular power in either extremity, and there was some wasting of both of them. The hernia cerebri grew gradually larger until it became tremendous in size and the skin over it in one place became necrotic and infected. On November 27, the necrotic area had extended so that it had involved a great part of the scalp over the hernia, and pus was freely discharged from sinuses in the hernia. On this day, the patient became comatose and died five days later.

REPORT OF AUTOPSY.—Performed by Dr. J. H. Globus, twelve hours *post mortem.*

Head.—Over the right temporoparietal region there is a large hernia cerebri, 17 cm. in long diameter, 14 cm. wide, 10 cm. deep. The apex of this hernia is very much flattened and presents a large necrotic surface with the skin over it sloughed away, disclosing a large mass of dead tissue with numerous pockets of pus. The necrotic tissue is of a grayish-green color containing dark brown hemorrhagic areas in the depths. At the margin of this tumor where it joins the skull, marked induration and infiltration of the subcutaneous tissue is found. Here, also, one can trace the encapsulating connective tissue of the tumor attached to the margin of a window cut out in the temporal region of the skull. From there this connective tissue covering the free surface of this mass can be traced to the dura. The window above mentioned measures 8 x 6 cm., and is the result of a decompression operation.

Brain.—The brain proper is of normal size. Left hemisphere presents no abnormalities. The gyri are flattened and the fissures are shallow. The right hemisphere presents a striking picture. A large mass of the dimensions described above is found attached by an extremely broad peduncle on the dorsolateral surface. This peduncle covers an area which stretches over a large part of the frontal, temporal, and parietal lobes. This area includes the precentral gyrus and a narrow zone in front of it, the postcentral gyrus, the gyrus supramarginalis, part of the gyrus angularis, most of the superior parietal lobule, the posterior two-thirds of the superior temporal, the posterior half of the middle temporal, and the posterior tip of the inferior temporal. The peduncle consists of a mass of soft tissue, the center of which is extremely necrotic. On moving it aside, there is a large cavity, about 10 cm. in long diameter, 6 cm. in width, this cavity no doubt representing the lateral ventricle with all its subdivisions although the three horns of the lateral ventricle cannot be made out. Within what represents the descending horn there is a soft mass which has the outline and course of the hippocampus major. The large tumor supported by the above-described peduncle is made up of a thin capsule of soft white tissue beneath which large masses of necrotic tissue and circumscribed effusions of blood are seen. Several small pockets of pus are also recognized.

The Histological Features of the Neoplasm

Tumors derived from the supporting tissues of the brain and spinal cord are the most common neoplasms of the cerebrospinal axis, and are perhaps somewhat too frequently placed by clinician and pathologist alike in the large vague group of gliomata.

It was Virchow, soon after the discovery of neuroglia, the supporting framework of the central nervous system, who established the identity of many brain tumors, ascribed their derivation to the glia tissue and named them gliomata. He described such new growths as slow-growing, infiltrative, vascular masses in which, by virtue of their exceedingly high vascularity, hemorrhages occur. These readily lead to softening and cyst formation.

But while his macroscopic findings still remain the important, distinguishing gross anatomical characteristics of gliomata, his microscopic descriptions have suffered many changes in the light of recent researches and the discovery of the newer specific staining methods.

Without entering into a lengthy review of the gradual development of our present knowledge of the nature of gliomata, it will be well to summarize the views held at present as to the origin, structure, and character of gliomata, and other genetically allied brain tumors.

Gliomata are now defined as tumors, ectodermal in origin, and microscopically consisting of glial cells and extracellular glia fibers. They are found only in the central nervous system and its outgrowths, the great majority occurring in the brain. They vary in size from a minute nodule to the size of a complete hemisphere. They may acquire any shape, but are usually round in outline in the brain, and often assume the form of a capsule about the cord. They are usually solitary, metastasis being of doubtful occurrence. Stroebe (1) found in one case a small, well-defined, secondary and, in his opinion, metastatic nodule a short distance from the seat of the primary growth. Heubner (2) found in the spinal cord of his case a series of independent gliomata extending over an area of fifteen segments. Gliomata *rarely invade the brain coverings*, and never extend beyond meninges unless as the result of necrotic changes. They vary in color from yellowish-white to grayish-pink. They are subject to degenerative changes as the result of alteration in their vascular supply. Areas of softening and cyst formation are thus occasioned. Gliomata are of *slow growth*, extend by infiltration of the adjacent brain tissue, and form no definite line of demarcation between the normal brain tissue and the tumor mass, unless pressure and hemorrhages have led to the formation of a necrotic zone about the periphery of the neoplasm. As a rule, gliomata form soft masses, though, as in the case of fibromata, hard and soft gliomata are recognized. Such distinction depends mainly on the relative predominance of fibers or cells.

In describing the microscopic features of gliomata Mallory (3) says: "The cells in glioblastoma (name used synonymously with glioma) tend to differentiate into neuroglia cells, *i. e.*, into cells which form neuroglia fibrils. Whether they do that or not depends much on the rate of growth of the tumor. If the cells proliferate slowly, neuroglia fibrils may be formed in large numbers; if they multiply rapidly, few or no fibrils may be produced. Complete lack of fibril formation necessarily renders diagnosis doubtful." Tumor cells of glioma vary in shape and size. Their arrangement is also variable, they often give rise to cystlike structures lined with ependymal cells, often assuming glandular character and again may present a molecular arrangement as in sarcomata.

Highly cellular gliomata, because of their histologic make-up and clinical features, often bear a striking resemblance to sarcomata. Fibrils in such growths, due to rapid proliferation of their cells, are often absent or, when present, cannot be demonstrated because of unavoidable delay in fixation of material caused by circumstances incident to the obtainment and the performing of autopsies. In such instances diagnosis is often made extremely difficult, if not impossible. Such tumors are often erroneously described as gliosarcoma and even as sarcomata, for lack of a definite criterion which would enable one to assign a definite place to such a new growth.

Stroebe finds an important point of departure between gliomata and sarcomata in that, in the former, fibers are numerous and tend to form a reticulum, while fibers are few, if there are any, in sarcomata, and they never form a reticulum. He is inclined to believe, however, that there are mixed tumors, gliosarcomata, in which sarcomatous zones about blood vessels are alternating with true glial zones.

This simplicity of classification of gliomatous tumors as suggested by Stroebe is only apparent, for it leads to no radical solution of the problem and leaves a large number of atypical, transitional forms unclassified.

Borst (4) meets this difficulty by introducing the term gliomasarcomatodes for tumors which clinically bear all the ear marks of a sarcoma, but morphologically are indistinguishable from a highly cellular glioma.

The wide variation in the cellular make-up and cellular arrangement of such rapidly growing soft gliomata lead to the creation of many other new names. The presence of giant cells produced the name *giant-cell glioma;* tumors in which glandular structure was simulated resulted in the coinage of the name *adenoglioma*, while for tumor in which cyst-like structures are lined with ependyma-like cells the name *neuro-epithelioma gliomatosum* was adopted.

Kaufman (5) rightly states that this terminology is often misleading, and this is especially true of the contradictory term sarcomatous glioma. Gliomata being recognized as of ectodermal origin cannot be considered sarcomata, for the latter are by definition tumors derived from mesoderm. This difference in derivation does not permit of the idea that transformation can occur. To designate the rapid growth of such a tumor he introduces the name of *cellular malignant glioma.* For tumors rich in the columnar type of cells seen radially arranged about cystlike structures he suggests the name *columnar cell glioma* and provisionally the term *spongioblastoma.*

Such terms, descriptive as they may be, are nevertheless not based on any fundamental

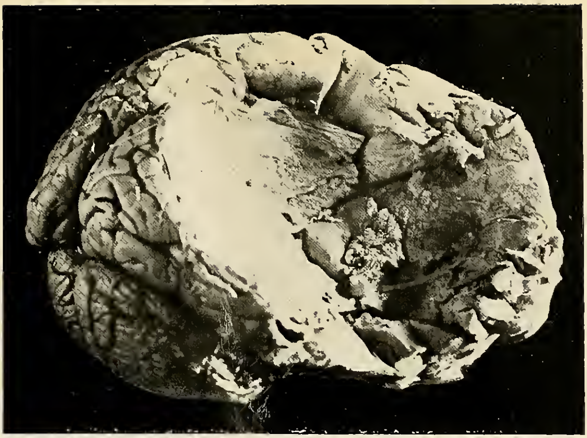


Fig. 1. Dorsolateral Surface of Right Hemisphere with Neoplasm Removed

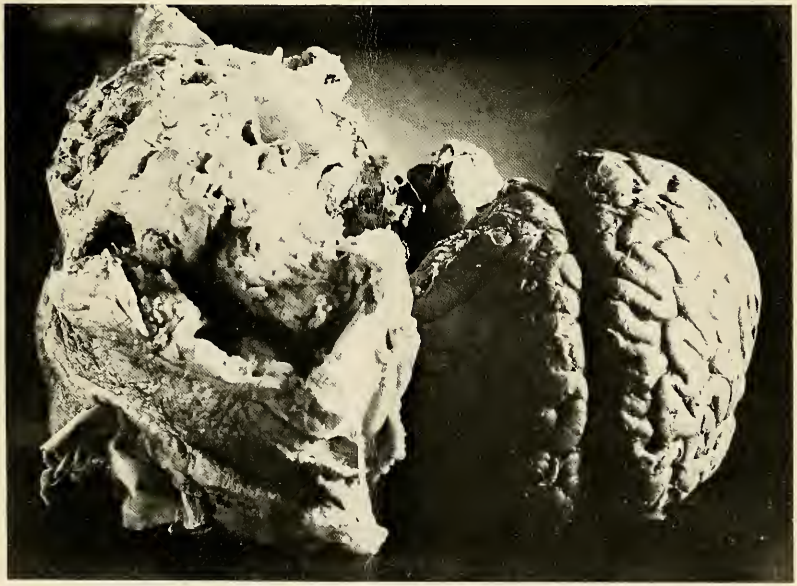


Fig. 2. Photograph Showing Relative Size of Brain and the Tumor Growth

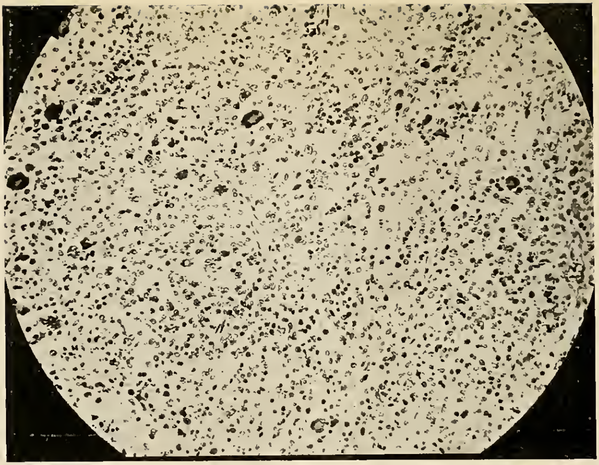


Fig. 3. The Histological Features of Neoplasm are well Shown here

Cystlike structures and numerous giant cells are seen throughout the microscopic field

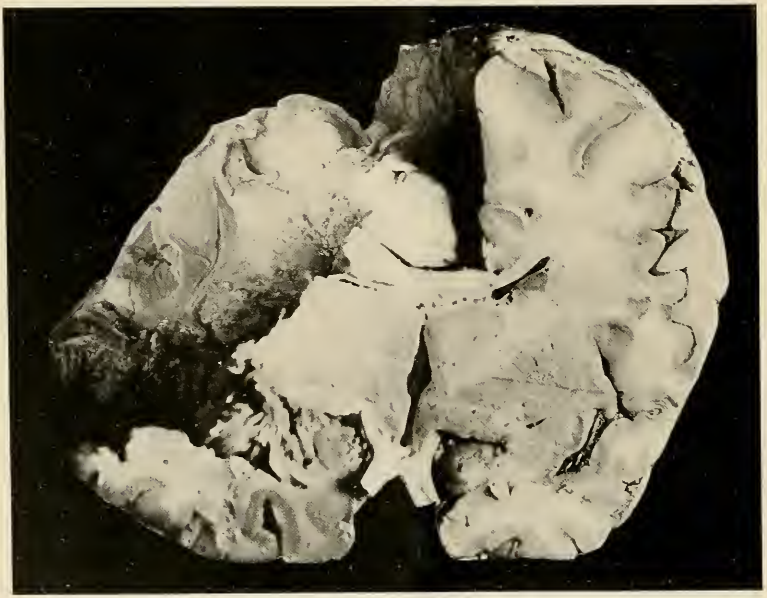
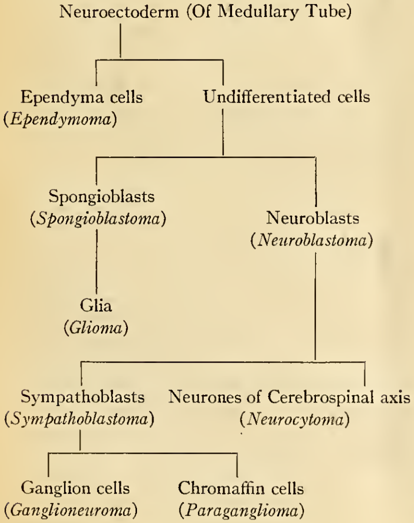


Fig. 4. Frontal Section of the Brain Showing Marked Destruction of Right Cerebral Capsule

morphological principles, but are merely adaptations to some apparent gross and microscopic features of the new growth. In spite of the evident confusion which exists in the classification of atypical gliomatous growths, no serious attempts were made to establish the basic histological and embryological factors governing the morphology and clinical behavior of atypical gliomata. Indeed, it is now well understood that true gliomata and true neuromata obtain their cellular elements from cells genetically related to the neural epithelium, which, when arrested in their development at some stage of differentiation and then suddenly caused to proliferate, form structures typical of a given stage in the histogenesis of the central nervous system.

Thus we have come to recognize tumors such as neuroblastoma, ganglioneuroma, sympathoblastoma and paraganglioma, and each of these characteristic new growths can be directly traced to such histogenetic elements as neuroblasts, ganglioblasts, sympathoblasts and the chromaffin cells.

The following diagram is introduced to indicate the various stages in the differentiation of the known specialized units of the central nervous system. It permits of a graphical presentation of the views held as to the origin of various neoplasms from specific nerve elements in the course of their differentiation. These views seem to be in full accordance with our knowledge of the histogenesis of the central nervous system.



The above schema shows the various stages the primitive neuro-epithelial cell undergoes before it gives rise to the highly specialized nerve units. Here we see that two distinct divisions occur early in the differentiation of the neuro-ectoderm; they are represented by the spongioblasts and neuroblasts. The *spongioblasts* are concerned with the elaboration of the specific supporting glia tissue of the central nervous system, while the *neuroblasts* are the forerunners of the neurons of cerebrospinal axis and the migrating sympathoblasts. The sympathoblasts leave the ganglion crest at an early stage of development and migrate ventrally, where they enter into the formation of a series of sympathetic ganglia in the region of the trunk. Certain of these ganglion cells do not form nerve cells, but acquire the character of peculiar, pigmented cells which are concerned with the elaboration of an internal secretion. They are the chromaffin cells, the constituent elements of the chromaffin bodies. Chromaffin derivatives of the celiac plexus later become engulfed by the mesodermal anlage of the adrenal and form the medulla of this gland of internal secretion.

Tumors whose component cells have been found to correspond morphologically to one or another stage in the development of specific nerve elements have been described. R. A. Lambert (6) collected from the literature twenty-four cases of malignant neuroblastoma, tumors derived from the undifferentiated neuroblasts. Neuroblastoma originate most commonly in the adrenal glands, growing rapidly, infiltrating the surrounding tissues and giving rise to numerous metastases. They occur most often in infants. Microscopically they are characterized by cells, small in size, with round nuclei, rich in chromatin granules, and in places, at least, these cells are associated with delicate fibrils which do not give the characteristic reaction of neuroglia fibres with specific stains. Some of the cells are pyriform in shape and the cytoplasm is prolonged into processes. Such cells are often seen arranged in a circle, with their delicate processes forming a network in the center which gives rise to a characteristic structure known as the *rosette.*

The true nature of this tumor was not recognized until a few years ago. Tumors originating in the adrenal and presenting strikingly similar features of the above-described neoplasm were variously reported as lymphosarcoma, round-cell sarcoma, glioma and gliosarcoma of the adrenals. It is quite certain that in the light of recent studies many of these were examples of neuroblastoma.

Another distinct and well-known type of tumor occurring in the retina, and often erroneously diagnosed as glioma, is now known to be derived from the specialized elements of the retina. It is characterized by formation of small glandlike cavities lined by cells, closely simulating the rods and cones of the retina. Such tumor was named *neurocytoma.*

Martius (7) in 1913, reported a tumor consisting of the sympathetic formative cells and named it *sympathoblastoma.*

Tumors containing ganglion cells in large numbers together with nerve fibers, the latter often being well formed and even medulated, have been described under the name of *ganglioneuromata.* They are rather rare, occurring in connection with sympathetic ganglia of thorax and abdomen. They may also occur in the skin, where they are believed to originate in the walls of blood vessels.

Finally, Wahl (8) considers the occurrence of paraganglioma theoretically possible. Indeed, it is quite true that with a working hypothesis based on embryological facts we shall be in position, in the near future, to establish the true identity of many neoplasms often believed to be atypical hypernephromata. We are now engaged in the study of a tumor clinically diagnosed as hypernephroma, which, however, is primary in the adrenal and presents many unusual features. It is extremely rich in chromaffin cells, making the diagnosis of hypernephroma somewhat doubtful.

It is very gratifying to note that with the growth of our knowledge of the histogenetic changes that take place in the central nervous system the employment of such data has led to the identification of many atypical neuromata, and saved many of them from being thrown indiscriminately into the "waste basket group" of sarcomata.

It is unfortunate that the classification of gliomata is not as satisfactory, although similar embryological factors are operative in the development of gliomata as in the case of true neuromata, tumors derived from the parenchyma of the central nervous system. The histogenetic basis for classification of gliomatous tumors is well supported by the fact that ependymal cells give origin to a class of neoplasm to which the term ependymoma has been correctly applied. But in the further differentiation of the embryonic ependymal cells there is a transitional form of cell, the spongioblast, which forms a link between the embryonic neuro-epithelium and the neuroglia cell. Since the neuroblast, a transitional type of cell-forming neurons, is capable of developing into malignant neuroblastoma, the spongioblast, in our opinion, can likewise form the basis for the development of a tumor which should be designated as a spongioblastoma. Such tumors were not described, however, often as they may have occurred. They, if recognized, would no doubt constitute a large percentage of those neoplasms frequently designated as gliosarcomata. Such, in our opinion, is the case in the sarcoma *molle* described by Erdman, a neoplasm strikingly similar in its clinical manifestation and microscopic structure to the tumor we are reporting, and which we consider to be a spongioblastoma.

In this tumor there are several characteristic and distinct types of cells. One dominant type of cell which apparently makes up the bulk of the neoplasm is, in our opinion, rather low in the scale of differentiation and shows striking similarities to the primitive neuro-epithelium. Though variable in size and shape, these cells bear a close resemblance to those so characteristic of neuroblastoma with their vesicular nuclei, deeply staining chromatic granules and, in many instances, pyriform shape of the cell outline, as shown in Fig. 6. Many of these cells are seen to give off blunt processes, while

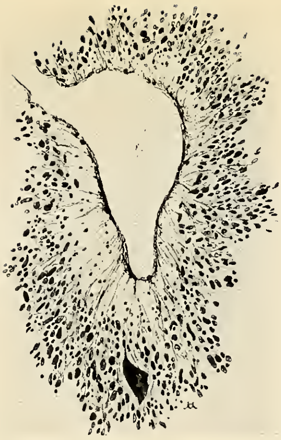


Fig. 5. Low Power Drawing

Showing the ependyma-like cells
arranged radially about a
blood vessel

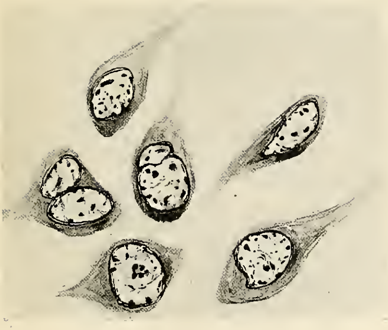


Fig. 6. The Dominating Type of the Tumor Cells

They are pyriform in shape, possessing one or more
short, blunt processes. Nuclei, vesicular, clear,
filled with deeply-staining chromatic granules

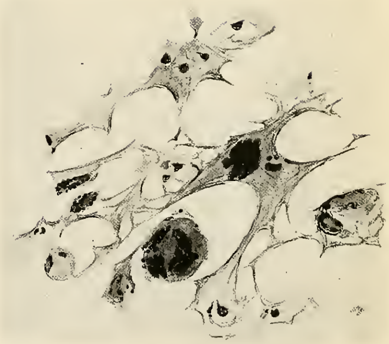


Fig. 7. High Power Drawing

Showing reticulum formed by interlacing and
anastomosing processes of spongioblasts
and giant cells

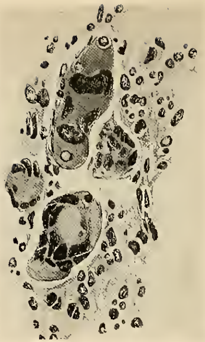


Fig. 8. High Power Drawing Showing Several Typical Giant Cells, Surrounded by Numerous Naked Nuclei

The latter are imbedded in a homogeneous protoplasmic matrix, acquiring the character of a syncytium

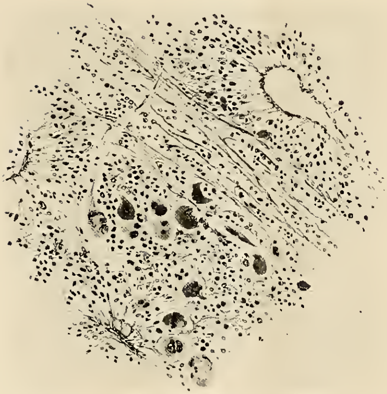


Fig. 9. Low Power Drawing of an Average Microscopic Field

The histologic features of the neoplasm are here very apparent. Blood vessels with radially arranged ependyma-like cells, numerous giant cells, and the highly cellular character of the new growth are here well shown others are brought in contact by means of these processes with neighboring cells, thus often giving rise to a reticulum as shown in Fig. 7. Round cells are also found in large numbers, the nuclei of which are surrounded by variable amounts of cytoplasm, and supported by a homogeneous matrix of a granular or reticular character, thus giving the appearance of a syncytium. There are also many cells which are pyramidal in outline with processes at one pole and with a full-rounded base. The latter are interesting, not so much because of their individual characteristics, as by reason of their peculiar arrangement, and also because of their relation to blood vessels and other spaces which appear to be cysts. The microscopic field is everywhere traversed by connective-tissue trabeculae, enclosing blood vessels. About the latter, tumor cells, conical in outline, are grouped in a radiating fashion. Their processes are directed in more or less parallel lines and run at right angles to the enclosed structures, *i. e.*, blood vessels and cysts. In this way they often appear actiniform, as shown in Fig. 5.

The giant cells, the other prominent type, are found in large numbers, equally and diffusely distributed throughout the microscopic field in many of the sections. They vary in size, outline and number of nuclei. Some are relatively small, containing but one deeply staining nucleus, while the majority are of rather large size, often containing as many as six to eight well-formed and well-defined nuclei. In some of these cells the nuclei appear to be gathered at one pole, in some the nuclear material is equally distributed at both poles, indicating a final stage in cell division, while in others mature, independent nuclei are arranged in the form of a ring at the periphery of the cell. Many are found with several nuclei in the center which overlap each other, or are connected by chromatic threads. The nuclei of this second type of cells are also of the vesicular type, clear and filled with deeply-staining chromatic granules. Mitotic figures are found here in large numbers as well as in the spongioblasts, *i. e.*, the above-described undifferentiated glia cells. The cytoplasm of the giant cells is variable in amount, assuming various forms, round, oval, stellate and pyriform, giving off blunt processes at the narrowed pole of the cell. The giant cells are evidently not phagocytic in character as is the case of giant cells occurring in granulation tissue or in inflammatory reaction of tissues, but they are large germ cells, rapidly undergoing atypical division.

A few small, isolated patches are found where extracellular glia fibers are recognized. Furthermore, throughout the section there were several well-circumscribed areas of infiltration with polymorphonuclear leukocytes alternating with hemorrhagic zones and areas of necrosis.

Thus, the histologic features summarized are:

1. The neuro-epithelial character of the dominating type of cells.
2. The presence of well-defined spongioblasts and ependyma-like cells.
3. The syncytial structure alternating with reticular interlacement of cell processes.
4. The marked proliferative tendencies.

It is significant that, besides the very apparent resemblance of structure between neuroblastoma and spongioblastoma, we also find marked similarity in some of their clinical manifestations. Both tumors are malignant in that their growth is very rapid and in their tendency to infiltration. The rapid growth is directly traceable to the low degree of differentiation of their component cells. They differ, however, in their capacity to form metastases. The neuroblastoma are capable of rapid and widespread metastasis and are thus highly malignant. In such a tumor we are dealing with cells that are potentially migratory, while the spongioblasts destined to become supporting glia cells are anchored to the cerebrospinal axis.

Several cases of multiple "sarcomatous" gliomata have been reported, but the metastasis, if such occurred, cannot be considered as examples of true metastasis, for the multiplicity here was limited to the cerebrospinal axis.

DISCUSSION

(After the case was generally discussed in clinic, the following specific points were amplified.)

Dr. Strauss.—I should like to ask Dr. Tilney where he would transplant these gliomata. In human beings?

Dr. Tilney.—Not necessarily.

Dr. Strauss.—Biological conditions demand that we transplant these tumors in the same type, the same animal. I have on four occasions taken gliomata removed at operation and gone at once to the animal room and either injected a suspension of the glioma or transplanted pieces into the cortex of Macacus rhesus after destroying the pia, and there always was absorption. I doubt if gliomatous tissue would live in another species.

Regarding the causation of this condition, someone brought up the question of trauma in brain neoplasms. I called attention to the fact that this woman had headaches before she sustained the blow. It may be that the headaches were the first symptoms of a latent neoplasm. The blow either caused hemorrhage in the neoplasm near the internal capsule, or it stimulated the growth of these cells. If these cells are those of a glioma they are derived from epiblast, and trauma may have influenced their growth as it does epitheliomatous structures elsewhere, as on the lips of the pipe-smoker and the tongue or cheek from irritation of a tooth. Likewise, it is possible that there are some individuals in whom trauma interferes with the inhibition which holds pathological tissues in check; instead of getting an ordinary scar they develop a keloid type of scar. There must be something in those individuals to produce that reaction. Trauma is not always followed by the formation of sarcoma.

As to the question of Dr. Sharpe in reference to the operation, it was not a subtemporal decompression; a large osteoplastic flap was made over the supposed site with the idea of going in subcortically and removing the neoplasm.

About the thickening of the bone, it was directly due to the growth. The thickening which was found at the site of operation is some form of irritation caused by bone hypertrophy which occurs occasionally in cerebral neoplasms. The neoplasm may be in the center of the brain.

I would like to ask Dr. Holden a question. Does glioma of the eye make metastasis of the body? Because, if so, it is entirely different from the glioma of the brain.

Dr. Holden.—Glioma of the retina differs entirely from that of the brain. You may get metastasis anywhere from retinal glioma.

Dr. Strauss.—Glioma of the brain has never been known to do more than infiltrate the pia mater; it never forms metastasis. All of you have had this experience: in performing a decompression you generally look for one or two years of life in that individual. That is the average length of time the individual with brain tumor will live after relief of intracranial pressure. Glioma can remain in the brain for a long period of time without causing death. I know of one case in which the first symptoms took place five years before death. Even on the day of his death the man was downtown in the stock exchange; on his way home he was seized with transitory weakness of the legs, but was able to proceed home, a distance of twenty blocks, and walk up the stairs to his room, where he was found unconscious and died in an hour. I had the opportunity to examine the brain at once and found a tremendous glioma in the right temporoparietal lobes. A large hemorrhage had occurred in it and had ruptured into the ventricles. The first symptoms were manifested five years before. He had been seen by many specialists, and the consensus of opinion was that the patient was a neurasthenic. The unusual thing about the case I presented today was the rapid growth and development of this tumor in four months, and its growth outward. However, Dr. Tilney's assumption was not correct; it did not grow through the skull, it met with subcutaneous tissue and then with the dura.

Dr. Tilney.—May I ask a question in technique? When you operate and are certain you have a large tumor, though it is not visible on the surface, in the matter of needling the brain, how extensively should that be carried out? How safe is it?

Dr. Strauss.—There is one precaution which should be observed in aspirating the brain. The surgeon should never use a sharp-pointed needle. He should either use the needle suggested by Cushing or one with a blunt point containing a stylet. I have seen serious hemorrhage occur in the brain and the ventricle from the use of a sharp needle. I do not believe in indiscriminate and repeated punctures. There must be a reasonable suspicion of the existence of an abscess or a cyst, and the localization of the lesion ought to be fairly definitely established on clinical grounds. It is indefensible to blindly stab the brain in various directions with the hope of striking something. In cases where there is a suspicion of an abscess and the puncture by needle does not yield pus, the surgeon should follow the puncture pathway with a grooved director. He is even justified in making an incision into the cortex and inserting a small dressing forceps, because occasionally the pus is so thick it will not flow out except through a fairly large opening. I must, of course, again warn against any of these procedures unless there is a reasonable certainty both as regards the existence of an abscess and its location. For purposes of diagnosis, you can use a small calibred blunt needle to which a syringe is attached. By suction as the needle is withdrawn, small plugs of tissue are obtained which can be prepared and studied microscopically. In this way we made certain of the existence of the tumor in the case we have presented, although it was located subcortically.

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A CASE OF HEMIPLEGIA WITH HEMIANESTHESIA ALL QUALITIES OF SENSATION INVOLVED

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NEW YORK CITY

Patient: K. C. Female, aged 42, single, American. Admitted to the Presbyterian hospital May 2, 1918, complaining of paralysis of the left side.

Family History.—The mother died of heart trouble at fifty-five years of age, the father died of pneumonia at forty-eight years. There is one brother living who has sciatica and one sister who is in good health. There is no history of any chronic disease in the family.

Personal History.—The patient was born in New Jersey and has lived there during her lifetime. She has always eaten regularly. She drinks two cups of coffee daily, no tea or alcohol, and uses no drugs or mediums of any kind. Works in an office and has very little out-of-door exercise. Her average weight is 131 pounds; there has been no recent change.

Previous History.—When a child the patient had measles, mumps, chicken-pox and whooping-cough. One year ago she suffered a nervous breakdown, but prior to that her health had always been good. She has never been injured and has had no operations. Vision has always been good. She has never had headaches. She has occasional colds during the winter months, but no nasal abnormalities; has never had earache or any aural discharge. Her teeth are in good condition and have needed little repair. She has never had tonsillitis or been subject to sore throat. At no time has she had dyspnea, cyanosis or edema of the extremities. Her appetite is always good; bowels tend to be constipated. There has been no nocturia or diurnal frequency of urination. Menstruation is normal; periods commenced at thirteen and have always been regular, every twenty-eight days; duration four days; no dysmenorrhea.

One year ago she had a nervous breakdown. After working hard until eleven o'clock one night, she went to bed and slept soundly. The next morning she did not wish to get up. Finally, after arising, went to take a bath feeling very drowsy, but otherwise well. After taking the bath she was unable to get out of the tub for some time. After the water was drained out she was finally able to get back to her bed where she went to sleep and remained in a sound slumber until three o'clock in the afternoon. On awakening she felt very weak and was advised by her physician to remain in bed for three weeks. At the end of three months she felt sufficiently recovered to return to work, but since then has always been irritable and easily provoked.

Present Illness.—On May 1 the patient came home from work feeling rather tired and went out for a walk with her sister. They walked about twenty-four blocks and then came home and did some sewing. While sewing she noticed that a shade was not repaired which she had complained about many times to the landlady. This made her extremely angry, and after exhausting herself trying to fix the shade, she stepped down from the chair and immediately felt a numbness in her left arm. She rushed to the lavatory and let hot water run on the hand, believing that this would bring life back to her arm. At that time she could move her arm but not the fingers. She returned to her chair in great excitement and with a feeling as though her heart would burst. At this time she became confused and does not know exactly what took place except that she could not move her left side.

She was admitted to the hospital about twenty-four hours after the onset of the stroke. At this time she showed alternating periods of listlessness and restlessness, with a tendency to be childishly loquacious and to repeat the same complaints many times. Her chief complaint was abdominal pain due to "gas." Her speech was thick, but there was no difficulty in the selection of words.

There was considerable variation in the blood pressure during the first twelve hours after admission:

		Systolic	*Diastolic*
Blood Pressure 5/2 at	6:15 p. m.	215	125
	7:30 p. m.	220	120
	9:45 p. m.	235	130
	10:30 p. m.	175	100
	11:30 p. m.	195	105
5/3 at	9:00 a. m.	220	130

Throughout this time the pulse ranged from 54 to 66 and the respirations from 10 to 16.

Voluntary Motor System. *Attitudes.*—The patient is lying dorsally in bed with the left arm lying flaccidly relaxed across her abdomen and the left leg extended and motionless. The left half of the face is immovable, masklike and expressionless with the nasolabial fold obliterated and the angle of the mouth depressed. The respiratory expansion of the left side of the chest is less than that of the right, and there is a protrusion of the left half of the abdomen with the linea alba bowed to the right.

Gait.—Not tested, as patient is bedridden.

Coördination.—There is no disturbance of coördination on the right side. Fingers-to-nose, heel-to-knee, pointing and pass pointing tests are well performed. Dysmetria and adiadochokinesis are not present.

Skilled Acts.—There is no aphasia or apraxia. The patient is able to salute and feed herself with the right hand.

Reflexes:

Deep	*Right*	*Left*
Jaw	Present	Present
Pectoral	Absent	Absent
Biceps	Present	Very active
Triceps	Present	Very active
Radial	Absent	Active
Ulner	Absent	Active
Wrist	Absent	Active
Patellar	Present	Very active
Achilles	Absent	Active
Dorsocuboidal, ext.	Absent	Active
Dorsocuboidal, flexion	Absent	Absent

Superficial	*Right*	*Left*
Ciliospinal	Absent	Absent
Supra-umbilical	Present	Absent
Supra-pubic	Present	Absent
Upper lateral abdominal	Present	Absent
Lower lateral abdominal	Present	Absent

Plantar and its Modifications		
Plantar flexion	Present	Absent
Babinski	Absent	Present
Chaddock	Absent	Present
Oppenheim	Absent	Present
Gordon	Absent	Present
Schäfer	Absent	Present

Abnormal Involuntary Movements.—None present.

Muscle Strength.—The patient is unable to sit up from the recumbent position. There is paralysis of all the muscles of the left arm and leg and lower facial group. The left trapezius is paralyzed, causing inability to elevate the shoulder. There is weakness of the left orbicularis palpebrarum and frontalis, the left side of the tongue, and the respiratory and abdominal muscles of this side. The palatal muscles on the left side are also paralyzed.

Muscle Status.—The muscles of the left arm and leg are hypertonic.

Abnormal Associated Movements.—Hulbronner's sign of flaccidity, the trunk-thigh sign of Babinski, the complimentary opposition sign of Grasset, Gaussel and Hoover, the hyperkinesis sign of Claude, the sign of Marie-Foix and its modifications are all present.

Nerve Status.—Nothing noted.

General Sensory.—There is an entire absence of the sense of touch, pain, temperature, pressure, posture and stereognosis on the left half of the body, except on the plantar surface of the toes and anterior half of the sole of the left foot. In this latter region the sensations of touch, pain, temperature and pressure are normal.

The left buccal mucous membrane is anesthetic to touch and pain, as is also the left cornea.

Cranial Nerves and Head. *Olfactory and nose.*—The sense of smell is normal on each side.

Optic and Oculomotor Apparatus.—Vision is normal; visual fields are normal; the discs are normal. The right pupil is 3 mm. in diameter and the left is slightly larger. The pupillary accommodation is normal. The palpebral fissures are equal, each measuring 10 mm. There is no exophthalmos or enophthalmos, each eye measuring 18 mm. with the exophthalmometer. Ocular deviations are absent and conjugated movements are normal. There is no nystagmus.

Trigeminus and Mouth.—The sensations of touch, pain, temperature and pressure are absent on the left side of the face and the corresponding buccal mucous membrane is anesthetic to touch and pain. The normal reflex is absent on the left and present on the right side. The supra-orbital reflex is present on both sides. All the movements of the jaw are normal and equal on the two sides.

Facial and Face.—There is complete paralysis in the inferior facial region and a slight weakness of the superior facial on the left side, as the left eye cannot be closed as tightly as the right and the left eyebrow is slightly lower.

Acoustic and Ear.—Normal.

Glossopharyngeus and Vagus, Pharynx and Larynx.—Subjective and objective taste is normal. The palatal reflex is present on the right but absent on the left side. Swallowing is slightly difficult, but this is probably due to the palatal paralysis.

Spinal Accessory.—There is inability to elevate the shoulder on the left side, but the actions of both sterno-mastoids is normal.

Hypoglossus and Tongue.—The tongue deviates slightly to the left.

Vasomotor and Trophic.—The temperature of the paralyzed and normal sides is not markedly different, although the peripheral portions of the left extremities are somewhat cooler than the corresponding parts on the right side. There is slight swelling of the left hand, and excessive secretion of sweat in both the left arm and leg. There is acute decubitus in the sacral region of the left side.

The pulsations in the left carotid seem to be greater in amplitude than those on the right, and the patient complains of severe sensations of throbbing pulsation in the left eyeball and in the left side of the face and neck.

CRANIAL MORPHOLOGY.—Normal.

SYSTEMIC STATUS. *Tegumentary.*—There is no abnormal pigmentation. Hair and nails are normal.

Cardiovascular.—The heart is normal in outline; there is a slight systolic murmur at the apex. It is normal in rate and rhythm.

Respiratory.—Normal.

Gastro-intestinal.—Normal.

Genito-urinary.—Normal.

Skeletal.—Normal.

LABORATORY TESTS. *Blood Count.*—R.B.C. 4,700; W.B.C. 12,800; Polys. 84 per cent; S. Lymph. 7 per cent; L. Lymph. 7 per cent; Trans. 2 per cent; Hg. 65 per cent.

Blood Wassermann.—Negative.

Blood Culture.—Sterile.

Blood Urea.—Three grams per liter.

Phthalein.—First hour 36 per cent.
Second hour 13 per cent.
Total 49 per cent.

Spinal Fluid.—Perfectly clear, normal pressure. Cell count–Nine lymphocytes. Globulin–Negative. Culture—Sterile. Wassermann—Negative.

Urine.—Specific gravity–1008. Reaction acid. Albumin, trace. Sugar, none. Microscopic, occasional hyalin and finely granular casts.

DISCUSSION.—The location of the lesion is, I believe, in the internal capsule. A cortical lesion would not be apt to produce so uniform and complete sensory motor symptoms. A cortical lesion on the right side in a left-handed person would probably produce dyspraxia of the right side, and the sensory disturbances would be apt to be limited to the correlation of sensory impulses, while the individual qualities of sensation might be unimpaired.

Considering the high blood pressure and the onset of the apoplectic attack during an emotional outburst, it seems to me that a cerebral hemorrhage is the most probable etiological agent.

TUMOR OF THE POSTERIOR CRANIAL FOSSA

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This case is of interest because of the difficulty in diagnosis and also because of the erroneous diagnosis of hysteria on the first admission to the hospital.

Patient: Female, 13 years of age, American. Admitted to the Presbyterian Hospital January 19, 1918, complaining of stomach trouble, headache, vomiting and inability to lie on the right side for four weeks. The mother and father are alive and well, also, four brothers are alive and well. There is no history of cancer, tuberculosis, kidney trouble or heart trouble in any member of the family.

Personal History.—Patient was born and has always lived in New York City. She has always had a regular time for meals and has a good appetite; does not drink tea or coffee. Her bowels are regular. Goes to bed at eight or nine o'clock and gets up at six; she sleeps well during the night. Her general health has always been good; she has never had any acute infectious diseases nor any operations. Six months ago she weighed 92 pounds (dressed), at the present time she weighs 70 pounds (undressed).

Head.—For a long time she has had persistant, severe headaches, the pains being most severe in the frontal region. Her ears, eyes, teeth and throat are negative.

Respiratory.—She has had a slight cough for two weeks, no sputum, no night sweats, no hemoptysis, no pain.

Cardiac.—No precordial pain, dyspnea or swelling of feet.

Gastro-intestinal.—Until the onset of the vomiting four weeks ago she has had no symptoms. The genito-urinary system is negative.

Menses.—Began during the past year. She has had four periods, each of five days' duration, regular at monthly intervals.

Present Illness.—The patient does not know the exact time of the onset of the headaches, but they have been severe for six or eight weeks. The pain is worse in the left frontal region, but radiates through the entire head. The vomiting commenced four weeks ago and has continued to the present time; it usually occurs about ten minutes after eating, is projectile in character and not accompanied by nausea. At first the attacks were infrequent, but later increased so that they occurred after each meal. The vomitus consisted of undigested food and never contained blood. There has been no epigastric pain.

For the past two weeks she has been unable to lie on the right side because it induces dizziness and severe headache. This position also causes a sensation of rotation, the objects about her moving from left to right.

Walking has become difficult because of a feeling as if she were going to fall, although she has never fallen. There has been no noticeable weakness in any part of her body.

Physical Examination. *General Appearance.*—The patient is a poorly developed and poorly nourished young girl. She has a pasty color and lies in bed with an apathetic expression, paying very little attention to anything that goes on around her.

Skin.—There are many comedones over the face and there is an acneform rash on the chest and back. The superficial layer of the skin is rough and dirty. There is very little fat anywhere. The hair is normal with an abundant pubic growth.

Superficial Lymph Nodes.—Cervical and inguinal glands are slightly enlarged.

Head.—Normal in size, shape and symmetry.

Eyes.—Pupils are equal and regular, react to light and accommodation. Conjunctivae pale, corneae anesthetic.

Nose and Ears.—Nothing pathological was noted on external examination.

Mouth.—The tongue is slightly coated; teeth carious, no pyorrhea. Pharynx is not infected or inflamed but sensibility is diminished. The tonsils are enlarged.

Neck.—Is not stiff and there is no thyroid enlargement.

Thorax.—Is small and both sides move equally with respiration. Breasts are small, conical and contain no masses. The lungs are normal. The heartbeat at the apex is not seen or felt. The outline is normal, the left limit of dullness being 9 cm. from the midline in the fourth interspace. The sounds are regular and of good quality and there are no murmurs. The pulses are equal, regular, and of fair quality easily compressed. The vessel walls are not thickened.

Abdomen.—The wall is thin and flat, no tenderness, rigidity, masses or signs of fluid. The liver and spleen are not palpable or enlarged to percussion.

External Genitals.—Nothing abnormal noted.

Extremities.—These are thin and wasted. There are no other abnormalities. Reflexes: Knee jerks are equal and active. There is no Babinski and no clonus.

Rectal Examination.—The cervix is small and firm. There is no tenderness or abnormal masses.

Temperature is 98.8; Pulse 92; Blood pressure 105–70.

LABORATORY FINDINGS. *Blood Count.*— Hb. 80 per cent; R.B.C. 4,300,000; W.B.C. 7,000; Polys. 51 per cent; S. Lymph. 26 per cent; L. Lymph. 16 per cent; Large Monon. 4 per cent; Basoph. 1 per cent.

Blood Wassermann.—Alcoholic antigen—negative; Cholesterin antigen—negative.

Stomach Contents.—Fasting—20 cc. of yellowish fluid, some mucus, no blood. Total acid 42; hydrochloric acid 26; no lactic acid.

Stool.—No blood, ova or parasites.

Lumbar Puncture.—Fifteen cc. of clear fluid, two cells. Wassermann reaction negative. Guinea pig inoculation gave no signs of tuberculosis.

X-ray of chest showed no indication of any disease of the lungs.

Urine.—Sp. gr. 1030; acid, amber, slight granular precipitate; very faint trace of albumin, no sugar. Microscopic examination. Few squamous epithelial cells and occasional white blood cells.

The patient was discharged from the hospital on January 25, 1918, with a diagnosis of hysteria.

SECOND ADMISSION to the hospital on May 18, 1918.

Interval History.—Since leaving the hospital in January, the patient has been bedridden. She has gradually been losing strength and has been unable to sit up in bed for more than an hour without complete exhaustion. The frontal headaches have continued and have increased in severity. These come on generally during the morning, occasionally at night, are of a dull, aching character and last about an hour. Lately she has had continual aching pain in the occipital region which is aggravated markedly by movement of the head. There has been no vomiting since she was discharged from the hsopital. There has been a marked impairment of vision amounting to nearly total blindness in the right eye and an inability to read ordinary print with the left.

GENERAL EXAMINATION. *General appearance.*—Inspection shows a poorly nourished girl of thirteen lying dorsally in bed with no evidence of pain. There is no deformity except for a slight internal strabismus of the right eye. The skin is dry and harsh and shows no abnormal pigmentation. She appears rather dull and uncommunicative and is not interested in what is going on about her. If interrogated, she answers slowly and with a weak, monotonous voice. Her memory is good and there is no evident impairment of intelligence. The rest of the general examination, except for some points noted in the following examination, is the same as recorded on her former admission.

VOLUNTARY MOTOR SYSTEM. *Attitudes.*—The patient lies in the dorsal position or partially on the right side with her head rotated to the right and inclined slightly forward so that the chin approaches the right shoulder. The attitude is rarely altered either during the day or night.

Gait.—The patient is scarcely able to walk and has difficulty in standing. In attempting to walk the feet are held apart and on making one or two jerky, lurching movements, she staggers and falls to the right. This incoördination is apparently uninfluenced by closing the eyes.

Coördination.—(a) Equilibratory—On attempting to stand with the feet together, there is marked swaying and irregular movements of the whole body with a tendency to fall to the right. This is not increased by closing the eyes. She is unable to stand on either foot alone.

(b) Nonequilibratory—Finger-to-nose test is accomplished with slight waving movements of the arm but is not very perfectly performed. Pointing and pass pointing give the same result. Heel-to-toe and heel-to-knee tests are carried out with slight wavering and imprecision and are equal on the two sides. Heel-to-knee along shin shows only slight irregular or uncertain movements. Dysmetria—Drawing imaginary horizontal lines to a vertical one is fairly well performed. Adiadochokinesis is not present on rapid succession of pronation and supination.

Skilled Acts.—Speech is rather slow but otherwise normal. There is no apraxia, the patient being able to salute, tie a knot, feed herself and touch the right ear with the left hand.

Reflexes:

Deep	*Right*	*Left*
Jaw	0	0
Pectoral	Present	Present
Biceps	0	0
Triceps	Slight	Slight
Wrist	0	0
Radial	0	0
Ulnar	0	0
Suprapatellar	0	0
Patellar	Present	Present
Achilles	Present	Present
Dorsocuboidal flexion	0	0
Periosteal	0	0
Crossed periosteal	0	0
Ankle clonus	0	0
Patellar clonus	0	0
Superficial		
Upper lateral abdominal	Present	Present
Lower lateral abdominal	Present	Present
Plantar and its Modifications		
Plantar flexion	Present	Present
Babinski	0	0
Chaddock	0	0
Oppenheim	0	0
Gordon	0	0
Schäfer	0	0

Abnormal Involuntary Movements.—There are no tremors, no twitchings, no choreiform movements, athetosis or spasms.

Muscle Strength.—The muscle strength is everywhere below normal, but is equal on the two sides and seems nowhere disproportionate.

Muscle Status.—The muscles of the extremities are soft and relaxed. This hypotonia is evidenced by hyperflexion and extension of the extremities. The fingers may be hyperextended until they are at a right angle with the dorsum of the hand. The thighs may be flexed until the knees touch the shoulders. The hypotonus is general and equal on the two sides.

Abnormal Associated Movements.—None are present.

NERVE STATUS.—Nothing noted.

GENERAL SENSORY. *Touch.*—Probably normal. There seems to be an inability to discriminate between camel's-hair brushes of different lengths on the arms, legs and trunk.

Pain.—Apparently normal.

Temperature.—Normal.

Vibratory.—Normal.

Pressure.—Normal.

Muscle-tendon.—Normal.

Stereognosis.—Normal.

Barognosis.—No observation.

CRANIAL NERVES.—*Olfactory and nose.*—Normal (alcohol, violet perfume).

Optic and Oculomotor Apparatus.—Vision is practically absent in the right eye; very much diminished in the left.

Eye Grounds.—Right optic disc: Temporal third gray; margins above and below indistinct. The entire disc is slightly raised. Left optic disc: Papilledema about 2 diopters; a few retinal hemorrhages below the disc.

Pupils.—Dilated, regular; the right slightly larger than the left—right measures 4 mm. plus and the left 4 mm. Reaction to direct light is diminished in the right eye, normal in the left. The consensual reflex is present on each side. Both pupils react to convergence. The right eye is slightly more prominent than the left, the exophthalmometer reading being 17 mm. in the right eye and 16 in the left. Von Graefe and Stellwag signs are absent and there is no ptosis, the palpebral fissures each measuring 7 mm. There is a slight internal strabismus of the right eye and an inability to move it to the right; all other movements of the eye are normal.

Nystagmus.—Horizontal nystagmus in both eyes on looking to the left and in the left eye on looking to the right.

The winking and blinking reflexes are absent on the right and present on the left side.

Trigeminus and Mouth.—Normal.

Facial and Face.—Normal. Taste on anterior two-thirds of the tongue is normal.

Acoustic and Ear.—Normal.

Bárány Test.—Patient recumbent. Cold water in left ear produced a slight, coarse, vertical nystagmus in each eye for a duration of three minutes and ten seconds and a rotary vertigo to the right. Cold water in the right ear produced a somewhat finer and more rapid horizontal nystagmus in both eyes with the slow movement to the left, also right rotary vertigo. There was no nausea or vomiting.

Glossopharyngeus, Vagus, Pharynx.—Normal.

Spinal Accessory.—Normal.

Hypoglossus and Tongue.—Normal.

SYSTEMIC STATUS.—As given before.

LABORATORY TESTS.—*Blood Count.*—B.C. 4,200,000; W.B.C. 15,000; Hb. 70 per cent; Polys. 82 per cent; S. Lymph. 9 per cent; L. Lymph. 1 per cent; Trans. 3.5 per cent; Eosin 4 per cent; Basoph. 5 per cent.

Lumbar Puncture.—Fifteen cc. of clear fluid under greatly increased pressure removed.

Spinal Fluid.—Culture sterile. Guinea pig inoculation negative. Search for tubercle bacilli negative. Cell count, nine lymphocytes. Wassermann reaction negative.

Von Pirquet skin positive.

Blood Wassermann negative.

X-ray of chest.—No indication of disease of the lungs; very small heart shadow.

X-ray of skull.—Slight increase in density of both maxillary sinuses and both anterior ethmoids, turbinate bones very much enlarged. Cella turcica smaller than the average; clinoidal processes almost completely bridged over.

Urine.—Sp. gr. 1025. Acid, clear, no albumin, sugar or casts.

DISCUSSION.—The diagnosis of intracranial tumor seems reasonably sure, as the cardinal phenomena of this condition—headache, vomiting and optic neuritis—are present, and evidences of nephritis or lead poisoning are absent.

The rotary vertigo, the reeling gait, the hypotonia, nystagmus and attitude of the head, together with the early optic neuritis, point to localization in the cerebellum, though cerebellar asynergia is absent.

The blindness in the eye with the least papilledema is difficult of explanation on this ground, and rather tends to indicate direct pressure on the optic nerve by a tumor of the frontal region. Against this assumption is the slight improvement in vision after lumbar puncture, and the fact that a possible tumor in the region of the right optic nerve, causing blindness by pressure, would probably also implicate the corresponding olfactory nerve, which is normal in this case.

A CASE OF POSSIBLE ANTERIOR POLIOMYELITIS REVEALED BY SUBSEQUENT OCCUPATION*

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While the diagnosis cannot be regarded as established beyond doubt, this case presents points of interest on account of its rather atypical form and as showing how an unobserved poliomyelitis may lead to further complications.

The patient, M. G., Case No. 20,448, is a boy ten years of age. He is right-handed.

History. *Present Illness.*—The patient complains of pain in the left shoulder produced by pressure on the anterior aspect of the humerus a short distance below the head. The weakness brought out subsequently was not mentioned in the original complaint. The patient and his mother are both "easy-going" individuals, and apparently not given to observation of their ailments.

The pain was first noticed about six weeks before the patient presented himself in February. During the previous October the boy had begun taking violin lessons and the pain was brought on by the pressure of the violin against the left shoulder. He had difficulty in holding up the violin very long, but apparently at first this was not more noticeable than is usual in some beginners. He complained that he got tired, and was told that boys younger than he could hold it up. An older brother questioned the teacher on this point, and the latter said that the patient was apparently weak at the outset. The teacher did not notice any change in his condition during the ten weeks he took lessons. The brother thinks he has become weaker since, this increase in weakness being attributed by the brother to the arm having been strapped up three weeks to relieve any condition causing pain. The strapping was done by another department which had previously referred the boy to this department. On a second examination, subsequent to this, the patient did appear to give an impression of greater weakness; also it would seem that the weakness tremor developed in holding the arm extended (see below) would have been noticed if existing from the first. An increase in weakness, however, cannot be regarded as certain.

There has been no fever noticed in connection with the trouble. No cramps or colic, no dysesthesia or paresthesia, no pain except as complained of above and no cutaneous changes have been observed by the patient or his family.

Previous History.—The birth and development of the patient were normal. He had, chicken-pox, whooping-cough at three years, measles at five. An operation for adenoids five or six years ago is said to have been followed by a great deal of hemorrhage. Since then he is stated to have been very liable to colds and catarrh. He was confined to his bed several days last winter by influenza.

Family History.—This is negative.

Voluntary Motor System. *Abnormal Attitudes and Deformities.*—None were noted except a slight lordosis.

Gait.—Normal.

Coördination.—Normal except for tremor noted below.

Skilled Acts.—Normal.

Reflexes.—(a) Deep: These are all rather slight. They are present in the left arm, except the ulnar, which is absent in both arms, and a doubtful radial. The triceps is more marked on the right.

(b) Superficial: These are all present, not especially marked, but normal. There is no Babinski or clonus.

Abnormal Involuntary Movements.—There is a tremor of the left arm when held extended, evidently due to weakness. There are no fibrillary twitchings.

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* Presented at the Clinical Conference of the Neurological Department of the College of Physicians and Surgeons, April 22, 1918

Muscle Strength.—The following muscles show marked paresis on the left: the adductors, rotators, (especially supinators) and extensors (forward) of the arm, including the pectorals; the supinators and extensors of the forearm and the dorsal extensors, abductors and adductors of the hand.

The following show less marked or doubtful paresis, doubtful because the left, normally the weaker, extremity is in question: the flexors of the forearm and hand, and the flexors, extensors, abductors, and adductors (?) of the fingers. In the fingers flexion weakness appears to be more marked in the distal phalanges.

Muscle Status.—Volume: There is a diminution in volume of the left shoulder, apparently extending to the left scapular and interscapular region, the left arm, and forearm. The greatest diameter of the arm and forearm is one-half inch less than that of the right; right and left being respectively seven and a half and seven inches. This difference was the same before and after the strapping referred to above.

Consistency: The muscles are rather soft in both arms but more so in the left.

Tonus: There is diminished tonus in the left arm.

Electrical Reactions: There is diminished faradic excitability in the left deltoid, biceps, triceps, and forearm extensors and flexors, but no reaction of degeneration.

Abnormal Associated Movements.—These were not tested.

NERVE STATUS.—This was not investigated.

GENERAL SENSORY.—Touch, pain, temperature, vibratory, pressure and muscle tendon senses are all normal. At first it was thought there might be a slight hyperesthesia to pin prick and a slight hypesthesia to cold on the left shoulder and arm, but subsequent investigation did not confirm this. There is no tenderness on pressure except at the point mentioned above, and no pain on any active or passive movements of the hand, arm, or shoulder.

CRANIAL NERVES AND HEAD. *Olfactory and Nose.*—Not examined.

Optic and Oculomotor Apparatus.—The fundi, light reflexes, eye movements, and corneal sensibility are normal.

Acoustic and Ear.—Not examined.

Trigeminus and Mouth.—The jaw movements are normal.

Facial and Face.—Normal.

Glossopharyngeus, Vagus, Pharynx and Larynx.—These were not examined.

Hypoglossus and Tongue.—The tongue movements were normal.

MENTAL STATUS.—Not tested, but apparently normal. The patient is a restless boy; is said to have always been so.

CRANIAL MORPHOLOGY.—Nothing noted.

SYSTEMIC.—*Tegumentary*, *Glandular*, *Cardiovascular*, *Respiratory*, *Gastro-intestinal*, *Genito-urinary*, *Skeletal*.—These were not especially examined, but nothing abnormal was noted on superficial examination. The heart is normal. The bones of the left hand give an impression of being smaller than those of the right.

LABORATORY TESTS.—*Wassermann*, *Lumbar Puncture*, *Blood Count* and *Urine* were not taken. An *x*-ray of the left shoulder and one of the neck showed nothing abnormal excepting an area of possible rarefaction about one-half inch below the neck of the humerus, possibly a periostitis. (Subsequent *x*-rays showed the presence of a periostitis, apparently inactive.) A von Pirquet test was negative. (A subsequent von Pirquet developed apparently a violent reaction).

DIAGNOSIS.—The possibilities suggested by the findings are: (*a*) brachial neuritis, (*b*) anterior poliomyelitis, (*c*) myopathic amyotrophy, (*d*) chronic neuritic amyotrophy, (*e*) chronic nuclear amyotrophy, (*f*) spinal cord neoplasm, (*g*) occupation neurosis. Occupation neurosis may be excluded by the atrophy, as there was not sufficient disuse nor was it of such a distribution as to be thus accounted for. The fact that there is no inability to perform any particular skilled movement, but a genuine paresis of certain isolated muscles is also against this diagnosis, although the force of the latter consideration may be somewhat diminished by the fact that the neurosis in this case would take the form of an inability to hold an object in a certain position rather than to perform an active movement. Neoplasm affecting the cord or roots is excluded by the lack of sensory and tract symptoms. Neuritis appears to be excluded by the lack of sensory symptoms (excepting the pain on local pressure), both objective and subjective, in both the present condition and past history. There is also no ascertainable cause for a neuritis of this form either traumatic or otherwise, including an arthrogenous form, there being no arthritic symptoms. The possibility of a partially recovered neuritis should, however, be kept in mind.

The *kind* of disturbance rather favors a myopathic amyotrophy, but the distribution is not of any of the more usual types. Myopathy, moreover, may be excluded on account of the unilateral character of the trouble. Possibly more time will be required to make this point absolutely decisive.

The unilaterality also militates against a chronic neuritic and a chronic nuclear amyotrophy, especially the former. In addition to this, the lack of sensory symptoms is against the former and the lack of fibrillary twitchings excludes the latter.

The form of the muscular involvement fits in very well with the arm form of anterior poliomyelitis, but against the latter is the absence of the reaction of degeneration, the retention of the deep reflexes in the affected area, and apparently some evidence that the weakness has progressed. There is also an absence of any definite history of an illness with paralysis and this disease is at present rare in this locality. These objections are diminished by assuming that we are dealing with a light case in which there might be no reaction of degeneration and a retention of the deep reflexes, though it must be admitted that the latter is still a serious objection. The lack of anamnesis and improbability of occurrence are more readily explained by assuming that the trouble is not recent, but may have occurred during the last epidemic. The patient and his family, it must be remembered, were not apparently aware that there was any weakness before the clinical examination. On the other hand it must be remembered that the weakness would have been greater and more noticeable at the onset.

In view of the preceding, the case is admittedly difficult and is one depending upon relative probability values of various symptoms, values which are difficult to estimate with precision. Nevertheless it would seem that the probabilities at present favor anterior poliomyelitis.

According to the view offered, then, the patient suffered, some time ago, probably—possibly in the last epidemic—from a light attack of anterior poliomyelitis which, as it involved only the left arm, was not noticed. Any accompanying constitutional disturbances may have been attributed to some ordinary complaint. When, however, unusual work was required of the muscles involved in holding the violin, the weakness became apparent and was possibly increased by overfatigue. It is interesting to note that even then it was not the weakness but pain on pressure that attracted the patient's attention. The pain, in my opinion, was caused simply by too continued pressure upon a rather poorly protected part, though a neurotic element may here be present. Even if additional data reveal a more serious bony lesion it is difficult to see how one of such a limited character could cause the muscular atrophy and paresis in question.

DISCUSSION

Dr. Charles A. McKendree.—While this case seems to me to be one of very mild anterior poliomyelitis, I believe we should eliminate the possibility of a bone lesion before coming definitely to the conclusion that this is an atrophy of the poliomyelitis type. I do not think the radiogram is particularly clear with regard to any lesion in the head of the humerus; two pictures, one of the right and one of the left shoulder, might be of more value. I do not know how to account for the tenderness over the bone, whether it is a bursitis or a lesion within the bone substance. The atrophy might well be caused by an effort on the part of nature to protect the joint from pain. But the picture on the whole seems to suggest poliomyelitis.

Dr. Sylvester R. Leahy.—I think the possibility of an atypical anterior poliomyelitis might be given consideration here. The mode of onset, the course and the atrophy are atypical, particularly the general atrophy involving the entire extremity. I do not think the area of rarefaction shown in the radiogram would account for the atrophy. A blood Wassermann and lumbar puncture might throw some light on the subject, though the patient does not present symptoms referable to a specific condition.

Dr. Randal Hoyt.—This case is similar to one I saw in the Clinic last summer, that of a girl who came in with atrophy of the left leg and later on was found to have a streptococcic abscess of the bone itself. She had an atrophy not very different from that of this boy. We have here atrophy and a point of tenderness, and it seems to me our attention should first be focussed on the obvious factors of the case rather than directed to a remote possibility. There is an atrophy that goes with the condition such a tenderness would cause, and the radiogram shows definitely an area of rarefaction.

Dr. O. S. Strong.—I cannot agree with the suggestions that the atrophy and weakness are referable to a possible local bony lesion of the humerus. The lack of proximity to nerve trunks, the lack of arthritic indications and the presence of atrophy in the scapular, forearm and even hand regions, to my mind, practically exclude this. I think the bony condition, whether secondary or independent, has no etiological value in explaining the atrophy.

SACRAL TABES OR CEREBROSPINAL SYPHILIS*

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This case is presented because after repeated examinations and prolonged observation it still offers a problem for diagnosis.

Patient: L.B.; fifty years of age; male; married; white; Austrian; butcher.

History. *Present Illness.*—On September 12, 1917, the patient came to the clinic complaining of pain in both legs, the left more than the right. The pain was described as constant, day and night, at times drawing and pulling, at others shooting from shin to instep and often up to the left hip and spine. Occasionally he gets paroxysmal cramps in both legs, more often in the left. Several times daily he has severe pain in the rectum, lasting ten to fifteen seconds.

The history dates back to 1903 when the patient suddenly felt pain in the left hip, due as he thinks to lifting a heavy weight. The pain, which was of a drawing character, lasted three weeks. For the next three years he experienced pain on lifting heavy weights. In 1906 he began to have constant pains, shooting in character and going up and down the left thigh into the leg. In 1912 similar pains set in in the right thigh, knee and leg. In 1914 he noticed loss of power in the left foot, and at the same time began to have pains in the rectum. This pain was paroxysmal, very severe and often kept the patient awake at night. In 1910 he began to have bladder disturbances. He always felt like passing water and had a burning sensation on urination. Since 1914 he has had occasional urinary incontinence at night. At times there was difficulty in starting the stream. Sexual power has weakened since 1912, that is, he had premature ejaculations; complete impotence since

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* Presented at the Clinical Conference, March 4, 1918

1916. He has had severe constipation since onset of illness.

The patient never had headaches. Vision: he says that since 1910 he has been seeing double but that his glasses correct that. Since 1913 he has had jerking pains in the left side of the face which is constantly sweating. He has a peculiar feeling as of ants crawling in the left shoulder and a numb feeling in the left buttock. The left foot always feels cold and on walking it feels as if he is stepping on a sponge. He feels unsteady in the dark and on washing his face. He thinks that his speech has been getting difficult of late, his tongue seeming to get stuck on pronouncing the letter S. The patient was told on several occasions that he had sciatica and inflammation of the hip. His habits are good; he takes no alcohol, coffee and tea moderately, no tobacco. Sleep is poor on account of the pains. Appetite is poor. He has lost fifty pounds since the onset of his illness.

Previous History.—This is practically negative except for a chancre in 1893, for which he received no treatment.

Family History.—Father died of some "nerve" trouble at 67; mother living, 82. One brother died of dropsy. No hereditary factors in ascendants or collaterals. He has three children living and well and his wife miscarried three times.

On examination, September 12, 1917, the diagnosis of tabes was made because of the following signs: Argyll-Robertson pupils, probable Romberg, absent knee and ankle jerks. The presence of a left drop foot and atrophy of legs was not considered against the diagnosis. The subsequent course of things, cursory re-examination and negative serological findings led to doubts as to the original diagnosis, and on March 3, 1918, the following complete examination was made.

Voluntary Motor System.—Left drop foot. No abnormal attitudes or deformities on standing or sitting. Gait shows slight pelvic phase, steppage left leg owing to the need of lifting it on account of the drop foot. He is somewhat unsteady in walking with eyes closed; cannot walk on toes or heels. Fournier's tests are normal except for unsteadiness on turning abruptly. Coördination: There is some swaying in all directions with eyes closed (Romberg?). Finger-to-nose and heel-to-knee tests are normal. There is no adiadochokinesis. Skilled acts are all normal.

Reflexes:[1]		
Deep	*Right*	*Left*
Jaw	1	1
Pectoral	1	1
Biceps	2	2
Triceps	1	1
Radial	1	1
Ulnar	1	1
Suprapatellar	1	1—
Patellar	2	1+
Achilles	1+	1
Periosteal (crossed and uncrossed)	0	0
Superficial	*Right*	*Left*
Ciliospinal	0	0
Supra-umbilical	1	1
Suprapubic	1	1
Upper lat. abdom.	1	1
Lower lat. abdom.	1	1
Cremasteric	1	2
Anal	0	0
Plantar flexion	0	0
Babinski	0	0

No abnormal involuntary movements observed. Muscle strength: Patient cannot rise from recumbent position with hands folded on chest. Dorsal flexion of left foot and toes impossible; slight weakness of dorsal flexors of right foot. Plantar flexion of left foot greatly diminished; slightly diminished in right. Normal contour of legs. Atrophy of left leg and thigh, two and three centimeters respectively less than right. Loss of galvanism and faradism in dorsal flexors of left foot.

Nerve Status.—Normal.

General Sensory.—Touch, pain, temperature, vibratory, muscle-tendon and stereognostic senses all normal, except as follows: Hypalgesia, hypesthesia and hypothermesthesia over penis, scrotum and an oval area around anus extending about four inches laterally and a little below gluteal folds, corresponding roughly to fourth and fifth and possibly third sacral. Possibly similar diminished sensibility over external malleolus of left leg.

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[1] (1) signifies slight, (2) active, (0) absent.

CRANIAL NERVES. *Olfactory and Nose.*—Normal. Smell is normal.

Optic and Oculomotor, Trochlear and Abducens.—Pupils irregular, left oval, R. 5 mm., L. 4.5 mm. Both do not react to light but probably do to accommodation. Dr. Ward A. Holden is of the opinion that we deal with an Argyll-Robertson pupil in the last stages; Dr. M. J. Schoenberg believes that there is no response to accommodation. Visual fields and eye grounds are normal. Palpebral fissure is normal and so are the eye movements in all directions. There is no nystagmus.

Trigeminus Nerve and Mouth—Normal.

Facial and Face.—Normal, except that the patient constantly sweats on the left side. Taste is normal.

Acoustic and Ear.—Normal.

Glossopharyngeus, Vagus, Spinal Accessory, and Hypoglossus.—Normal.

MENTAL STATUS.—Normal.

CRANIAL MORPHOLOGY.—Apparently normal.

SYSTEMIC. *Tegumentary.*—Slight varicose veins on left leg. Constant sweating left side of face and forehead.

Glandular.—Cervical, axillary, epitrochlear and inguinal normal.

Cardio-vascular.—Blood pressure 120 systolic and 60 diastolic. Heart and arteries normal.

Respiratory, Gastro-intestinal, Genito-urinary and Skeletal.—All normal.

LABORATORY TESTS.—Blood Wassermann, negative; spinal fluid Wassermann, cells globulin and Lange negative. *X*-ray of sacrum apparently negative.

SUMMARY AND DIAGNOSIS.—We have a history of (1) paroxysmal pains of almost fifteen years' duration, at first in the left leg, then in the right; (2) incontinence, possibly retention, severe constipation; (3) paroxysmal pains in the rectum; (4) chancre in 1893. Examination in September, 1917, showed a probable A–R pupil, probable Romberg, absent knee and ankle jerk. Atrophy of left leg and left drop foot. Those observations were confirmed by two or three men. March, 1918, we found a doubtful A–R pupil, questionable Romberg, present but unequal patellar and ankle jerks, sensory changes corresponding to the fifth, fourth and possibly third sacral, position and vibratory sense present in feet and toes, sweating left side of face, left drop foot, negative serology of blood and spinal fluid. Finally, intensive treatment for six months showed absolutely no improvement.

Obviously a diagnosis of tabes was justified at the first examination; this becomes very difficult, though not impossible, at the last. Cerebrospinal syphilis suggests itself at once and the variability of the deep reflexes and the possible (?) involvement of the dorsal cord (unilateral sweating) might lend plausibility to that. But there seems to have been no doubt of an A–R pupil and many authors (Erb, Dejerine, Oppenheim, Uhtoff, Spiller and Camp) deny its existence in a pure cerebrospinal syphilis. The sensory changes suggestive of a conus lesion could very well go with a sacral tabes as with a cerebrospinal syphilis. The history is more suggestive of the first than of the second. A negative serology does not favor either side, but absolutely no response to very intensive treatment certainly points away from cerebrospinal syphilis. The present patellars and even Achilles jerks, as well as no Romberg might go with an extremely low tabes. The absence of other signs of syphilitic involvement of the central nervous system (ocular palsies, for instance, or other cranial nerve involvement) detract from the probability of its being cerebrospinal syphilis, and the fact that the condition has persisted for fifteen years and grown worse despite treatment further weakens such a diagnosis. Neither, however, can one make a positive diagnosis of tabes.

One more possibility presents itself. Do we deal with a gumma of the cauda equina or a tumor impinging both on the conus and the sacral roots? A negative serology and no response to treatment may well go with a gumma of so many years' duration. On the other hand a syphilitic or other neoplasm would have grown to greater proportions and given more than irritative symptoms in so long a time. It is difficult to decide, therefore, whether we deal with a tabes, cerebrospinal syphilis or gumma of the cauda.

DISCUSSION

Dr. F. MacCurdy.—As far as the radiogram is concerned, the picture was taken in the wrong position to show anything in the sacral region because it was out of focus. In my opinion, a new picture should be taken.

Dr. Norman Sharpe.—Above the shoulders this man's symptoms are those of tabes and below he seems to be a tumor case. You could hardly combine the two and say it is tabes with gumma. I am inclined to doubt the diagnosis of tabes nor do I think that a diagnosis of tumor can be made with any more certainty because, in this event, you would find in the spinal fluid an increase in globulin, especially if he had had tumor for fifteen years. It is rare to find a tumor around the caudal roots that, in fifteen years, does not grow to an enormous size. Such a one would give rise to many more symptoms than this man has. Tabes would be the most natural conclusion from the man's history, but there are so many indications that are not tabetic that this diagnosis is not tenable. Rather, I should favor a diagnosis of combined column disease.

Dr. Randal Hoyt.—Last fall when this case was given to me for demonstration some of the students noticed the patient's pupils. They reacted neither to light nor to accommodation; they were absolutely still. Another point a student brought out was the sweating on the left side of the face. At that time the patient had paralysis of the left leg, and, as we found an area of insensibility to pain, we were convinced he had a dissociation. The combination of the sweating, the peculiar pupillary reaction, and the sensory dissociation made me regard the case as one of syringomyelia. That diagnosis would also explain the fact that the paralysis of the leg extends into the anterior horn.

Dr. Charles A. McKendree.—I have a special interest in this patient because he was sent here to the Clinic by an orthopedist to see me. Dr. Wechsler examined him and reported the case as one of tabes, which diagnosis I conveyed to my friend the orthopedist.

The next time I saw the patient, as Dr. Hoyt wanted a case of tabes to demonstrate to the third year class, I sent this man to him with several others. Dr. Hoyt tried to elicit some of the fine points of tabes and they were not present. I made an appointment with the patient, on the fifteenth of November, and found what I considered to be an Argyll-Robertson pupil on each side. I then devoted myself to a sensory examination and called in Dr. Osnato to verify my findings. I thought the patient had a tabetic dissociation type of sensory disturbance. His areas of hypalgesia and hypesthesia seemed to me to be patchy and not limited to any nerve distribution.

Here is a man ill for fifteen years; he has a specific history with insufficient treatment; diminution of sexual power; rectal and bladder disturbance; eye muscle anomalies; Argyll-Robertson pupils; a variable Romberg and drop foot (due probably to thrombosis). Certainly one has to consider the possibility of low tabes, even though the serological findings have been repeatedly negative.

Dr. M. Osnato.—This is a very interesting case, but I do not think the diagnosis is quite so definite as some of the other men do. If there were no symptoms above the cauda equina or lumbar portion of the cord, you could explain this condition by a low tabes, even though the patient has no loss of position sense. But you must consider that he has symptoms in the first dorsal level and first cervical segment of the cord. An old tabes with adhesions between the cord and the dura would produce a sacculation and cause a negative blood and cerebrospinal fluid to occur. But in this case the disease is not limited to the lower part of the cord. You have symptoms at the first dorsal level and, then again, you must explain the Argyll-Robertson pupils. If they are Argyll-Robertson pupils, the condition must be one of two things and it may even be one of three: syphilis, alcoholism and multiple sclerosis—if you admit that Argyll-Robertson pupils are found in multiple sclerosis. Syphilis must be considered, but just what form of syphilis would give this trouble I do not know. You might think of gumma in the cauda equina which would explain the terrific root pain, but in fifteen years you would have more than just limitation to the lower end of the cord. This man's reflexes were gone at one time and today they have returned; there was a difference in the ankle reflexes which has also disappeared. Also at one time the pupils undoubtedly seemed fixed, but now react to accommodation. There has been improvement in some of the symptoms and this occurs in multiple sclerosis. There have been changes in the sensory symptoms objectively, also; the diffuse lesions of various parts of the cord and the midbrain could be explained by that process. The sensory changes were not limited, as they are now, but were more extensive. In my examination, it seemed to me the man had touch preserved irregularly over the analgesic area, and this patchy distribution and uncertain nature of the sensory changes would go with multiple sclerosis. The atrophy in the left leg and the pain need not absolutely militate against a multiple sclerosis and, in my opinion, this diagnosis should be at least considered here.

Dr. M. J. Schoenberg.—An Argyll-Robertson pupil is a small one that does not react to light but does to accommodation; however, the pupils which do not react to light and have only a trace of accommodation are not Argyll-Robertson pupils, there being only one stage missing when they are not going to react either to light or accommodation. This patient has some oculomotor disturbance and due to this he has no convergent pupillary reaction. On examining the eyes before using cocaine, I found the left pupil was larger than the right and the right interpalpebral fissure was larger; the right eyeball looked exophthalmic. In Argyll-Robertson, cocaine has to be used for a long time, sometimes an hour, before there is dilation. This man's pupils dilated very freely and quite promptly after a few drops of cocaine, and they are also equal. If there was present a paralysis of the sympathetic, the pupil on the right side would not have dilated as much as the pupil on the left.

This is certainly a complicated condition. These pupils look characteristic of cerebrospinal syphilis. The man has not an internal ophthalmoplegia for he can read small print with his corresponding correction. The pupils as they are look as if the condition were spinal lues, not tabes.

Dr. I. S. Wechsler.—I am afraid I cannot agree with Dr. Hoyt in his opinion regarding syringomyelia. Dr. Osnato's remarks on multiple sclerosis, it seems to me, hardly fit this case, if we but consider the total absence of signs and symptoms that are found in multiple sclerosis and the persistence of such violent root pains. That an Argyll-Robertson pupil is said to occur in multiple sclerosis is very true, but it is extremely rare, and in order to make sure it belongs to the multiple sclerosis, one ought first to be positive of the diagnosis. Of course, Dr. Osnato is not positive of this. Dr. Sharpe's criticism strikes me as being rather pertinent, but I fear I do not see why he should introduce combined column disease when we have enough trouble already. Dr. McKendree has taken up the cudgels in favor of tabes, and perhaps I ought to agree with him seeing that before I was beset with doubts I, too, made the diagnosis of tabes. Finally, while Dr. Schoenberg has apparently proved there is no involvement of the sympathetic going to the pupil, he has not explained the unilateral sweating nor resolved our doubts as to the diagnosis.

A CASE SHOWING THE CLINICAL MANIFESTATIONS OF PROGRESSIVE LENTICULAR DEGENERATION

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The patient in whom the diagnosis of progressive lenticular degeneration is ventured would fall into the group of cases described by Wilson as those of the chronic type. In the series of thirteen cases reported in *Brain*,[1] there were two cases reported by Homén originally in whom the duration was six and seven years respectively and of whom the ages at death were nineteen and twenty-six respectively. The fact that our patient is still alive after nearly five years of illness need not deter us from making such a diagnosis. That she was seventeen years of age at the time of the onset of her illness should not be held to militate against the probability that this was a genuine case. The average age of Wilson's cases at the time of their onset was fifteen years. In one patient the onset was twenty-six. The fact that our patient is a sporadic case is not a vital argument against it, because in five of the original thirteen cases, there was no familial history. Of course, the diagnosis can only be definitely made at autopsy. Several men, Wilson included, have made positive diagnoses during life which have not as yet been confirmed by autopsy. Our purpose in presenting this case is that, since there is a good chance of getting a post-mortem, we will then have an opportunity to confirm our diagnosis. Those who have not recently read Kinnier Wilson's original article should bear in mind that he has frequently stressed the point that in these cases there is a great variability of symptoms. Our patient at one time was so spastic that she could not get up and had to be moved practically as one piece, but latterly she lost a great deal of this rigidity and finally improved so that she could be transported for presentation at one of the clinical conferences. This fact need not cast suspicion upon the case. Another point which may help in clearing up this case, clinically at least, is to call attention to the only pyramidal tract sign which is present; that is, the clonus which exists on the left side. This is a very recent symptom and has been fairly constant of late. Some men do not regard clonus as a particularly important sign of pyramidal tract destruction or irritation if it is the only sign present. However this may be, it is certain that despite the efforts of Wilson to place the disease of progressive lenticular degeneration in the class of definite extrapyramidal conditions, it may be possible that he has been too severe in this respect. One of his cases at autopsy showed distinct lesions in the internal capsule. A clinical finding in this particular case was what Wilson described as a doubtful extensor plantar big toe response. A perusal of this case makes it fairly clear that Wilson had probably found a true case with a definite Babinski, but like all enthusiasts with an idea to fix or illustrate, he has failed to admit even to himself that mild pyramidal symptoms may occur. At any rate, keeping in mind the fact that a number of autopsied cases have shown actual cavity formation—the cavity replacing the lenticular nucleus entirely in one case—there is no reason to believe that it is not possible to get mild pyramidal tract signs in a pure case of Wilson's disease from pressure of the fluid in the cavity on the internal capsule. A final point in this introduction to our case is the interpretation which may possibly be given to the rectal and vaginal hemorrhages which have occurred. In the case of the former, particularly in the absence of evidence of hemorrhoids, it is possible to cast suspicion on the liver. The liver does not appear to be enlarged and its lower border cannot be felt. In the case of the latter, this patient illustrates what Wilson commented on in his cases a number of times, namely, the disturbances of menstruation. Regarding this and another symptom which is also present in our patient, namely, sialorrhea, Wilson says that these are suspicious evidence of disturbances of the glands of internal secretion.

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[1] *Brain*, Vol. 34, part 4, 1912.

HISTORY. *Present Illness.*—The patient, a woman, aged 22 years, single, having previously been perfectly well, about five years ago suddenly fell to the floor striking her back on a table. She was carried unconscious to the bed bleeding profusely from the vagina. It is said that the patient passed large clots of blood and that the hemorrhage lasted several hours. After this time the hemorrhage ceased completely, but she lay in bed and was so extremely weak that she could not move her head and had to be fed. Several days later when she finally began to talk and move a little, she complained of a severe pain in her throat and in the upper part of the chest. This pain was so intense that she was doubled up, her head and knees touching during the paroxysms. She still remained in bed, but gradually the pain subsided somewhat only to return at the end of several weeks with increased severity. She was taken to the Long Island College Hospital at the end of two or three months, and there it was said that she had a paralysis of the throat muscles "from spinal cord trouble, and that the pains were due to inflammation." Soon after this, she became drowsy and lay in bed for months without moving and in a semiconscious state. During this period, the pains in the throat and chest were not evident, but at the end of each drowsy period, they would return with increased violence, and during the attacks of pain, she would not be able to talk at all and was doubled up in a knee-to-chin posture, which was the only position in which she could obtain relief. During one of these attacks, she completely lost her voice and has never since regained it, having spoken not a word distinctly for more than four years. The dysarthria and dysphonia have remained stationary, there being no remission whatsoever. About the same time that she lost her voice, she complained of pain along the spine and began also to lose the power in the muscles of the lower limbs and finally became practically bedridden.

The tremor began four years ago. The grimace or spastic smile, which the patient has, began about two years ago. Of late, the patient has occasionally lost some blood from the rectum. On several occasions, her legs have become so extremely spastic that they have swelled considerably, although, as the patient's mother expressed it, "there was no dropsy and the skin did not pit." Until the patient finally became bedridden, she walked, as the mother says, as if she were "bouncing on rubber."

The mental deterioration has been very rapid. The mother says she is sure that until about two years ago the patient was mentally as keen as she ever was.

Previous History.—The patient's mother states that the pregnancy as a result of which our patient was born was perfectly normal and uneventful, and that the labor was easy. The child walked at one year and progressed satisfactorily, was bright at school and left when she was a little more than thirteen, because a cousin who brought her up thought it best that she go to work. She worked as a messenger girl in a dry goods store for several years and then stayed home to keep house for her mother.

Family History.—The mother is forty-five years of age and is neurotic; she also suffers from headaches and metrorrhagia. The father died of tuberculosis at the age of thirty. He was a shiftless man who would not work. The mother finally deserted him, and after his death married again. She had three children with her first husband, of whom the patient is one, the other two being a girl, twenty-six years of age, and a boy, twenty years, who are both said to be physically and mentally normal. She has had no children by her second husband. She states, further, that there is no history in any of the branches of her family of any illness such as exists in our patient. She knows very little about her first husband's family and is not even sure that he died of tuberculosis, although she heard that he died from some wasting disease.

VOLUNTARY MOTOR SYSTEM. *Attitudes.*—The patient is fairly well nourished and sits rigidly, her hands lying in her lap, usually one crossed over the other. The fingers are flexed at the proximal joints and extended at the distal joints. The thumbs are pressed into the palms, the distal joint flexed and the thumb somewhat extended. The head is held rigidly straight between the shoulders and the neck stiff. Her face is often fixed in a grimace or grin. Her movements are made very slowly because of the stiffness and tenseness in the muscles.

Gait.—The patient walks with a spastic gait with complete absence of the normal associated movements of the upper extremities. Her legs are rigid and the knees flexed but little. Her arms are held stiffly by her sides, her head erect, and in fact, she gives the appearance of a walking automaton. Walking accentuates the tremor. At times, instead of progressing normally, she will bounce on the tips of her toes for all the world like a rubber doll. This, however, is usually when she is playful. In sitting or lying down she falls into her chair or lies down in bed awkwardly. At one time during the period of observation she was practically bedridden. She could not get out of bed without being lifted up and then came to an upright posture, her whole body tense and rigid. She was lifted up out of bed as if she were one whole piece. Later, however, this condition of spastic-like stiffness ameliorated and she resumed her usual method of progression.

Coördination.—On account of the great spasticity and lack of coöperation, examinations to bring out the coördinating status are not always satisfactory, but nonequilibratory tests, namely, the finger-finger, finger-to-nose, knee-to-heel and knee-heel tests, seem to be well done, if one disregards the action tremor which is made worse by these tests.

Skilled Acts.—These are very poorly performed, and speech is extremely difficult. There is dysphonia and dysarthria, although at times she can speak one or two words and will hum after a fashion. The voice is guttural and no other sounds are possible except these—no labials, linguals or dentals. The patient suffers so from her tremor that she is unable to write. She can perform all ordinary skilled acts such as dressing and undressing herself, and so forth. These are interfered with only by the tremor and spasticity. If the patient is coöperative and in the humor, she will obey complicated commands, both written and spoken, fairly well, and will perform purposive acts without difficulty.

Reflexes.—All the reflexes are equal and active, both the superficial and deep. On account of the great spasticity, it is impossible to bring out clonus, although tapping of the patellar reflexes is sufficient to cause an extremely marked response. (Of late, clonus has appeared on the left side with some easily exhaustible clonus on the right side). The superficial reflexes, including the abdominals, are present and equal, although the test at various times is interfered with by the rigidity with which the abdominal muscles are held. For this reason, also, it is difficult to say whether a Babinski is present at these times. It has been easy to examine this patient's plantar reflexes only during sleep, and on a number of occasions when this was stealthily done at night, there was the normal flexion of the big toe and no Babinski was elicited. There was no Oppenheim and no Chaddock. The abdominals were present and equal during sleep. Pupillary reflexes are equal and prompt, both as regards light and accommodation, although there is a weakness in convergence, the left ocular movements not taking part in this reaction and the eyeball seeming to remain fixed with the same degree of spasticity as the skeletal muscles.

Abnormal Involuntary Movements.—When in complete rest, the tremor is observable and highly suggestive of the paralysis agitans type in the hands, but when the patient attempts to do something, she uncovers a generalized tremor, particularly marked in the hands and head and trunk. The tremor is, at times, rhythmical, has considerable force, and not very much amplitude and is very much increased by stress and emotion. At other times, however, the tremor is extremely irregular and arhythmical, has a wide amplitude with great force, particularly during stress, and will spread over the whole body. The patient seems able, with coaxing, to control the tremor and, at times, is able to stop it altogether. It is not exactly of the intention type, for the patient is able to drink out of a cup without spilling a drop. At the commencement of grasping the cup and just before she is able to do this, the tremor is quite marked, but as soon as she brings the cup to her lips, the tremor practically stops, and she is able to drink the contents without spilling any of it.

Muscle Strength.—This does not seem to be impaired. The tendo Achillis and the muscles of the wrist are held so rigidly as to almost constitute a deformity at times, and it requires some force to overcome the spasticity.

Muscle Status.—There has not appeared to be at any time real hypertonus when the patient lay recumbent in bed. The muscles themselves do not feel hypertonic. Nevertheless, at the various joints in the extremities, particularly the wrists and knees, there was so much stiffness that one felt that hypertonus might be the proper word to describe the condition. When the pseudocontraction was overcome by passive movements, the muscles resumed their normal consistency. The one exception to this rule appeared to be in the muscles of the neck, which were held continuously rigid. Even during sleep the muscles of the neck, particularly the sternocleidomastoids and trapezii, felt hypertonic. There were no muscular twitchings nor fibrillary twitchings. There was no myoedema. The mother has described a condition which existed for a number of months in the earlier stage of the disease in which the muscles of the legs were tense and hard. The skin over the legs did not pit. This condition has now completely disappeared.

Abnormal Associated Movements.—None. In walking, sitting, and so forth, the abnormal associated movements were absent. The patient moved as a mechanical toy.

NERVE STATUS.—Electrical reactions applied to the facial, ulnar and other large nerves gave the normal formula. Tapping the nerves or pressing upon them failed to bring out hyperexcitability. The peripheral nerves were not nodular or unusually palpable.

GENERAL SENSORY.—Touch, Pain, Temperature, Vibratory, Pressure, Muscle-tendon, Stereognosis, Barognosis.—There were no changes in any of these sensations.

With a patient in the present mental condition of this one, an accurate sensory examination is practically impossible. I have examined her numerous times and am well satisfied that there are no sensory changes of any kind. Dr. Leahy, who has examined her on a number of occasions and to whom I am indebted for several observations made with a view to checking up my findings, also agrees that the general sensory examination gives the normal reactions.

CRANIAL NERVES AND HEAD. *Olfactory and Nose.*—Normal.

Optic and Oculomotor Apparatus.—There is no nystagmus. The optic nerve is normal. Examinations of the fundi by several men agree as to this.

Trigeminal and Mouth.—The corneal reflex is active and equal. Sensory examination of the entire distribution of the fifth, including also the tongue and the inside of the mouth, discloses no abnormalities. There is no paralysis of the muscles of mastication. The jaw can be moved from side to side.

Facial and Face.—The seventh nerve is not affected excepting as the marked sialorrhea can be attributed to defects in this distribution. Nearly always the patient has to wear a bib in order to prevent wetting her clothes from the constant flow of saliva. This is a variable symptom, however. The forehead is smooth and free from wrinkles in repose. The face and the normal angles about the eyes and nose and mouth are flattened out much like the condition seen in paralysis agitans. Upon effort, it can be seen that there is no palsy in the seventh distribution, the patient closing her eyes, lifting her eyebrows, and moving her face without difficulty. The mouth is usually wide open, and, quite constantly, the lips uncover the teeth and her face discloses a wide stiff grin without other outward evidences of amusement. This grimace is the usual condition, although at

times the fixed smile relaxes considerably. When the face is fixed in a smile, the skin and the muscles covering the malar regions are tense and hard. There is tremor of the lips only when the patient is stressed or excited.

Acoustic and Ear.—(a) Cochlear: Normal. (b) Vestibular: Cold water caloric tests give the normal nystagmus and pass-pointing reactions. (c) Ear: Hearing, both air and bone conduction, is normal.

Glossopharyngeus, Vagus, Pharynx and Larynx.—The patient will open her mouth only after considerable coaxing and then not very widely. It has been extremely difficult to get a good look at her throat and the inside of her mouth. It is difficult, also, to get her to protrude her tongue, but by persevering we have been enabled, and I am sure Dr. Leahy agrees with me, to determine definitely that there is no paralysis of the muscles of the pharynx and larynx.

Spinal Accessory.—This is normal. There is no paralysis in the distribution of this nerve, although, as mentioned previously, the neck and head are rigidly held even in sleep.

Hypoglossus and Tongue.—There is no paralysis of the tongue which can be moved in any direction, providing the patient wills to do it.

MENTAL STATUS.—The patient is suffering from real dementia and has reached the level of the low imbecile type. She is extremely emotional, cries and laughs involuntarily, although the elated mood is more frequent and constant. She is, at times, very vicious and will scratch and bite. She leads a practically negative vegetative existence. The type of dementia is undoubtedly organic. The most characteristic feature is the complete indifference of the patient. She will sit for hours facing a window and even for days, if allowed to. She makes no move to do anything for herself and does not interest herself in the other patients, except occasionally to bite and scratch them, or when she is in better humor, to play and laugh immoderately at anything which they say to her. The usual attitude, however, is one of complete shutting out of the environment. She makes open efforts at invitation to sexual indulgence to any male who comes near her. She laughs immoderately with or without stimulus. The laugh is spasmodic, and it is noticeable that her whole body will tremble in association with the muscular efforts required to keep up the laughter. Less seldom she cries spasmodically. At times she will attempt to engage in conversation, but this is very rare. Very rare, too, are the episodes in which she will suddenly rise from her chair and run up and down the room attempting to hit her sides with her hands while doing so. She is so stiff, however, that sooner or later, as she runs, she will fall, if not prevented. She shows no more interest in her mother, who calls on her occasionally, than she does in anyone else. She will not read. She does not appear to have hallucinations or delusions. She is usually easy to handle. She makes no requests and her desires are very simple. She eats a great deal and would eat continuously, if allowed to do so. When one can arouse her sufficiently, it has appeared to me that her memory is fairly good. She makes no complaint of any kind.

CRANIAL MORPHOLOGY.—Normal.

SYSTEMIC. *Tegumentary.*—The skin is dry but not abnormally so. There is the proper distribution of hair and she has an abundance of hair on her head. The nails are normal.

Glandular.—Normal.

Cardio-vascular.—The heart and lungs are normal. The blood pressure is 120 systolic and 80 diastolic.

Respiratory.—Normal.

Gastro-intestinal.—Normal, excepting for the occasional hemorrhages from the bowels which, on a number of occasions, have been very severe. There are no visible or palpable hemorrhoids to account for this.

Genito-urinary.—Normal. There does not appear to be any fibroid of the uterus. She has had metrorrhagia on a number of occasions.

Skeletal.—Normal.

LABORATORY TESTS.—*Wassermann* of the blood is negative.

Lumbar puncture.—Spinal fluid Wassermann is negative, no cells, no globulin, and Lange's colloidal gold test is negative.

Blood count.—Not done.

Urine.—Normal.

X-ray of the skull does not show an enlarged sella turcica and is otherwise negative.

Comment.—Whatever this case turns out to be at autopsy, it seems that we are justified in saying that she exhibits quite probably an extrapyramidal condition. It appears important to make some interpretation of the pain which was so characteristic a feature of the symptomatology of the onset of this case. Taking it with other symptoms of unreserved and uncontrolled laughing and crying, it may be recognized as a possible thalamic symptom. Quite a large percentage of these cases which have come to autopsy have shown an involvement of the thalamus. It is true that this involvement is not particularly great nor nearly so striking in extent and character as that seen in the lenticular nucleus, but nevertheless it has been an almost constant feature.

STUDIES ON THE MORPHOLOGY OF THE GRAY MATTER IN THE BRAIN STEM OF VERTEBRATES BY THE RECONSTRUCTION METHOD

By RANDAL HOYT, M.D
Instructor in Neurology, Columbia University
NEW YORK CITY

Primates

THE CENTRAL GRAY SUBSTANCE FROM THE PYRAMIDAL DECUSSATION TO THE AQUEDUCT OF SYLVIUS (STUDY NO. 3).

In the previous studies of this series I have endeavored to emphasize the diagnostic significance of the anatomical structures under consideration. The central gray substance, however, has no such direct interest to the clinician, yet, forming as it does the "core" of the brain stem a conception of its appearance and general arrangement is essential in order to orient those nuclei which are of great clinical value and which will be described at a later date.

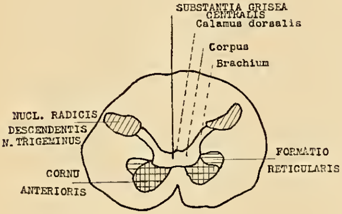
SUBSTANTIA GRISEA CENTRALIS
Calamus dorsalis
Corpus
Brachium
NUCL. RADICIS DESCENDENTIS N. TRIGEMINUS
CORNU ANTERIORIS
FORMATIO RETICULARIS

Fig. 2. Section 40

Fig. 1 is a diagrammatic representation of a detail in a reconstruction of the nuclear structures in the brain stem of a gorilla, illustrating the form of the central gray substance from the level of the pyramidal decussation to the aqueduct of Sylvius. The gracile and cuneate nuclei and the nucleus of the descending root of the trigeminus, are represented so as to show the dorsal and lateral relations of this substance. This diagram shows that this central gray substance has three natural divisions; a cylindrical caudal extremity; an expanded middle portion; and a cylindrical cephalic extremity. The cylindrical portions enclose the central canal and the aqueduct of Sylvius (as indicated by arrows). The

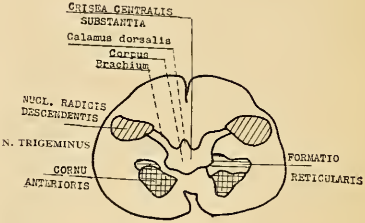

Fig. 3. Section 110

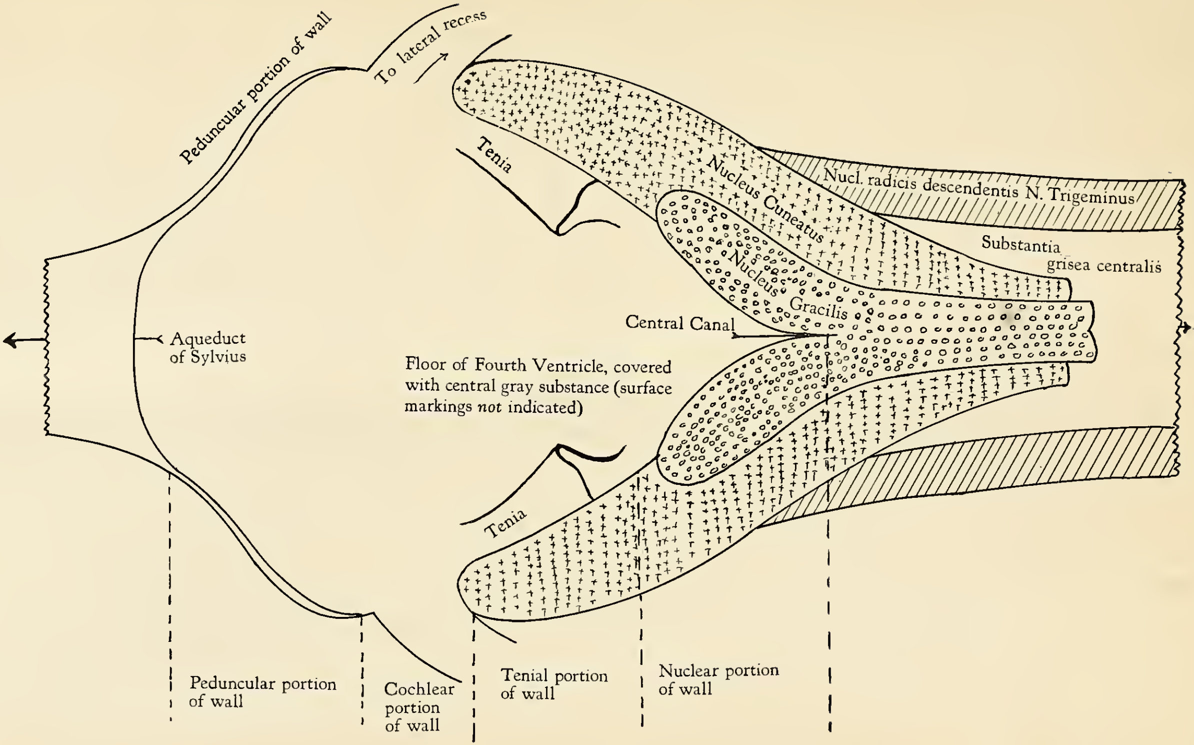
Peduncular portion of wall
To lateral recess
Tenia
Nucl. radicis descendentis N. Trigeminus
Nucleus Cuneatus
Nucleus Gracilis
Substantia grisea centralis
Central Canal
Aqueduct of Sylvius
Floor of Fourth Ventricle, covered with central gray substance (surface markings *not* indicated)
Tenia
Peduncular portion of wall
Cochlear portion of wall
Tenial portion of wall
Nuclear portion of wall

Fig. 1. Diagrammatic Outline of a Detail in a Reconstruction of the Nuclear Structures in the Brain Stem of a Gorilla

Showing the form and dorsal relations of the central gray substance

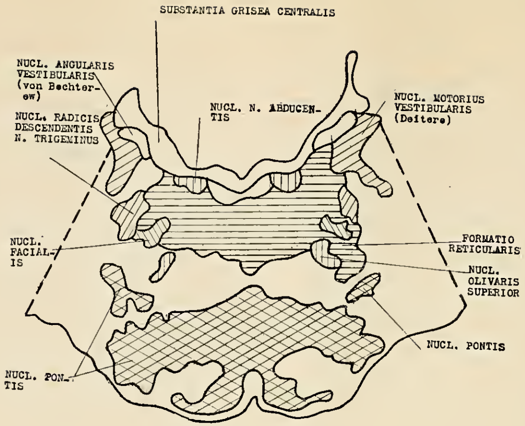
SUBSTANTIA GRISEA CENTRALIS
NUCL. ANGULARIS VESTIBULARIS (von Bechterew)
NUCL. RADICIS DESCENDENTIS N. TRIGEMINUS
NUCL. N. ABDUCENTIS
NUCL. MOTORIUS VESTIBULARIS (Deiters)
NUCL. FACIALIS
FORMATIO RETICULARIS
NUCL. OLIVARIS SUPERIOR
NUCL. PONTIS
NUCL. PONTIS

Fig. 11. Section 565

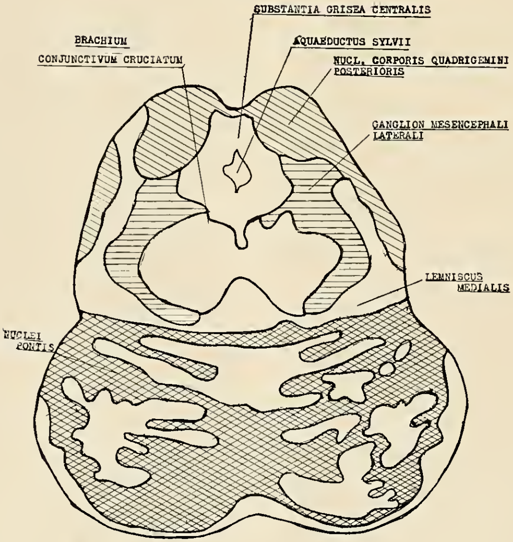
SUBSTANTIA GRISEA CENTRALIS
BRACHIUM CONJUNCTIVUM CRUCIATUM
AQUAEDUCTUS SYLVII
NUCL. CORPORIS QUADRIGEMINI POSTERIORIS
GANGLION MESENCEPHALI LATERALI
LEMNISCUS MEDIALIS
NUCLEI PONTIS

Fig. 12. Section 786

middle portion is open and forms the floor of the basin—the fourth ventricle. The caudal portion may be sub-divided, for purposes of description, into two parts—a spinal part and a bulbar part. The spinal part is the cephalic continuation of the central gray substance of the spinal cord and terminates at the caudal end of the gracile and cuneate nuclei. Its W-shaped appearance on cross-section, as outlined in Figs. 2 and 3, shows that it consists of a body (corpus), two dorsolateral arms (brachium), and a dorsal peak (calamus dorsalis). Comparing these structures in Figs. 2, 3, 4, and 5, it will be observed that the dorsolateral arms in Fig. 3 have become enlarged, that in Fig. 4 this enlargement has become "differentiated," and that in Fig. 5 the arms no longer exist but are replaced by large nuclear masses—the nuclei of the columns of Burdach (cuneate nuclei).

The term "differentiated" is used to express a peculiar feature of the central gray substance in regard to the formation of nuclei. Stained by the Weigert-Pal method, there are certain regions in which the central gray substance seems almost structureless: in other regions discreet darkly colored areas located within its boundaries will be seen. On closer examination, these areas are found to consist of a network of fine fibers and faintly stained cell-bodies. The fibers are the axones of the cell-bodies mentioned, or are the terminations of axones of other cell-bodies situated in a different portion of the nervous system. Such structures are termed "nuclei." As soon as such a nucleus is formed the central gray substance out of which it has developed ceases to exist; consequently there is a constant shifting of the peripheral outline of this substance, and a variation of its area in inverse proportion to the number of nuclei formed within it. It would almost seem as if the central gray matter acted as a "mother substance," functionless in itself, but capable of assuming dynamic properties as environmental adjustments demanded it. The nuclei of the columns of Burdach, in this manner, replace the dorsolateral arms of the central gray substance.

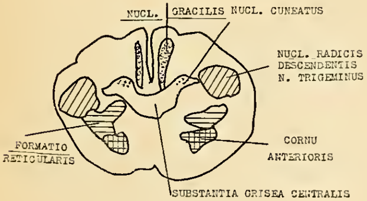
NUCL. GRACILIS
NUCL. CUNEATUS
NUCL. RADICIS DESCENDENTIS N. TRIGEMINUS
CORNU ANTERIORIS
FORMATIO RETICULARIS
SUBSTANTIA GRISEA CENTRALIS

Fig. 4. Section 160

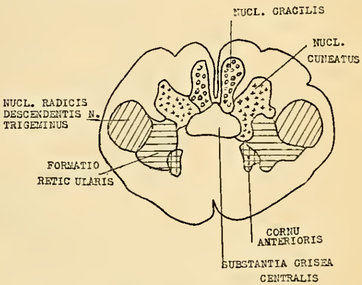
NUCL. GRACILIS
NUCL. CUNEATUS
NUCL. RADICIS DESCENDENTIS N. TRIGEMINUS
FORMATIO RETICULARIS
CORNU ANTERIORIS
SUBSTANTIA GRISEA CENTRALIS

Fig. 5. Section 230

The bulbar part of the caudal portion is concealed from view in the dorsal representation (Fig. 1), by the overlying nuclei of the columns of Goll and Burdach. In this region the relationship between the *dorsal peak* (Figs. 2 and 3) and the dorsomedian fissure is not represented in Fig. 2; in Fig. 3, it is a slit; in Fig. 4, it is a groove that separates the gracile nuclei, and in Fig. 5, it is shown touching the dorsal peak of the central gray substance. In Fig. 6, the dorsal peak extends into the dorsomedian fissure, and in Fig. 7, it has entirely disappeared, the body of the central gray substance remaining as the lateral boundaries of the now wide-opened dorsomedian fissure. The space thus formed is the caudal extremity of the fourth ventricle.

The body of the central gray substance does not occupy a constant position, but migrates dorsad. As evidenced by the reconstruction method, the structure that has the most fixed position in this region of the medulla is the nucleus of the descending root of the trigeminus, and, using this as a "landmark," it will be noted by comparison of Figs. 2–7, that there is a change of relationship between it and the central gray substance.

In Fig. 2, the body of the central gray substance is undoubtedly ventrad of the trigeminal nucleus, while in Fig. 7, it is definitely dorsad of it. Aside from change of position, the body of the central gray substance undergoes a change of shape which conforms to the increasing size of the gracile and cuneate nuclei, and to the internal arcuate fibers springing from them.

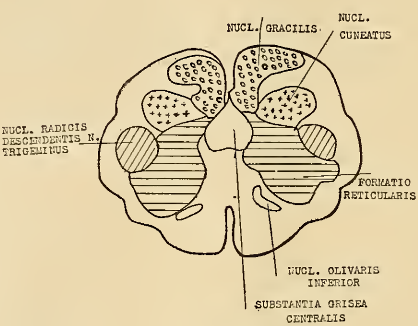

Fig. 6. Section 305

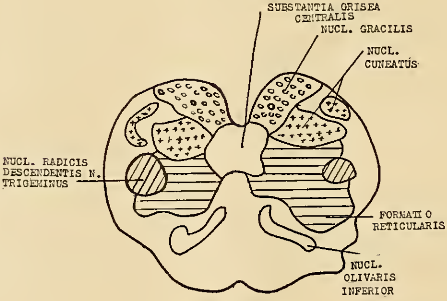

Fig. 7. Section 340

Soon after the dorsomedian fissure has opened, and has as its floor the body of the central gray substance, the central canal migrates dorsad and joins the opening thus formed. The lateral boundaries of this crevice are formed from the periphery inwards by the mesial aspects of the gracile nuclei and the central gray substance adjacent to the central canal. The central gray substance now spreads dorsally. It then increases in thickness and gives the appearance represented in Fig. 7. Cephalad to this region the central gray substance undergoes great differentiation into the nuclei of the ventricular floor, and for the reason mentioned above, its area is greatly reduced, as shown in Fig. 9.

It is not the purpose of this article to give a detailed description of the fourth ventricle, but rather to call attention to certain structures that the reconstruction illustrates. The dorsal extension of the central gray substance continues as a sort of fold or hooklike process, known as the teniae or ponticuli. The reconstruction of the teniae gives the appearance shown in Fig. 1. The ponticulus, therefore, acts as a lateral wall of the fourth ventricle in this caudal region. Following the teniae cephalad they again merge with the ventricular floor. At this point the cochlear nerve enters its nucleus and is located laterad to the restiform body (Fig. 10). As the nucleus enlarges

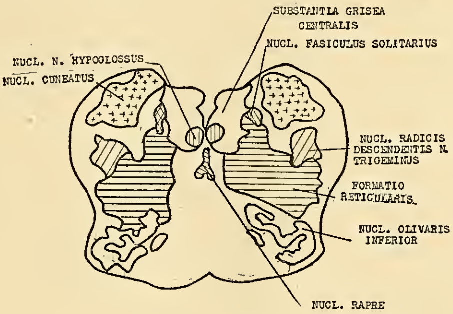
SUBSTANTIA GRISEA CENTRALIS
NUCL. FASICULUS SOLITARIUS
NUCL. N. HYPOGLOSSUS
NUCL. CUNEATUS
NUCL. RADICIS DESCENDENTIS N. TRIGEMINUS
FORMATIO RETICULARIS
NUCL. OLIVARIS INFERIOR
NUCL. RAPRE

FIG. 8. SECTION 365

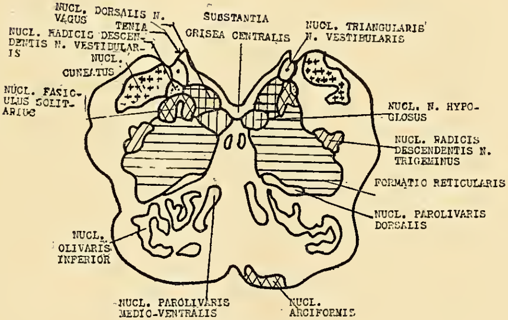
NUCL. DORSALIS N. VAGUS
TENIA
NUCL. RADICIS DESCENDENTIS N. VESTIBULARIS
NUCL. CUNEATUS
NUCL. FASICULUS SOLITARIUS
SUBSTANTIA GRISEA CENTRALIS
NUCL. TRIANGULARIS N. VESTIBULARIS
NUCL. N. HYPOGLOSUS
NUCL. RADICIS DESCENDENTIS N. TRIGEMINUS
FORMATIO RETICULARIS
NUCL. PAROLIVARIS DORSALIS
NUCL. OLIVARIS INFERIOR
NUCL. PAROLIVARIS MEDIO-VENTRALIS
NUCL. ARCIFORMIS

FIG. 9. SECTION 410

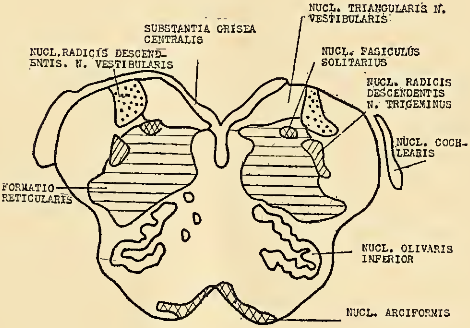
NUCL. TRIANGULARIS N. VESTIBULARIS
SUBSTANTIA GRISEA CENTRALIS
NUCL.RADICIS DESCENDENTIS. N. VESTIBULARIS
NUCL. FASICULUS SOLITARIUS
NUCL. RADICIS DESCENDENTIS N. TRIGEMINUS
NUCL. COCHLEARIS
FORMATIO RETICULARIS
NUCL. OLIVARIS INFERIOR
NUCL. ARCIFORMIS

FIG. 10. SECTION 480

it blends with the vestibular nuclei, which now occupy almost the whole region of the ventricle floor. Both of these nuclei are covered with a thin layer of central gray substance, which sweeps ventrally over the cochlear nerve (as indicated in Figs. 1 and 10). The fourth ventricle in this region has no lateral wall, but, on the contrary, extends ventrally so that the gray substance gives the appearance of falling over its edge. This lateral extension forms the mesial boundary of the lateral recess.

When the cephalic limit of the cochlear nucleus has been reached the restiform body is free to turn dorsad and pass into the cerebellum, thereby establishing a cephalic wall of the fourth ventricle, the remainder of this wall being completed by the juxta-restiform body and the superior cerebellar peduncle. The superior peduncles carry fibres from the dentate nuclei to the opposite red nuclei, and must therefore converge and eventually cross, and by so doing bring the walls of the ventricle to a point. These walls are lined with upward prolongations of the central gray substance, and when they at last meet the gray matter joins dorsally and encloses a canal, the aqueduct of Sylvius (Fig. 12).

The description of the peripheral outline of the central gray substance will be incorporated in the coming articles that will describe the nuclei of the ventricular floor.

NEUROLOGICAL BULLETIN

VOLUME I AUGUST, 1918 NUMBER 8

CEREBRAL GLIOMATA*

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NEW YORK CITY

The nervous system consists of tissue derived from the ectoderm; the nervous system proper, therefore, is of ectodermal origin. The ectoderm differentiates itself, first, into the neuroglia tissue, or the supporting framework of the nerve cells and fibers, and, second, into nerve cells proper. The most frequent tumors of the nervous system are the gliomata which arise from the neuroglia tissue. According to Cushing[1] they constitute more than half of all brain tumors. They are usually of an infiltrating type and have an indefinite boundary. They are, as a rule, highly vascular. Not infrequently, apparently normal looking and functionating nerve cells and fibers are seen running through them. Their size varies from that of a small marble to a growth occupying more than half of the hemisphere. These tumors arise within the brain and do not start from the meninges. Their increase in size, as a rule, is checked by the pia and they do not invade the tissue outside of the brain itself. Rapidly growing tumors ordinarily do not form glia fibers and are characterized by their highly cellular appearance. Only slow-growing gliomata differentiate fibers.

Characteristic features of these tumors are (1) a tendency to arrange themselves about the blood vessels in a radiating manner; (2) a tendency to produce glia fibers; (3) they do not form metastases; and (4) being highly vascular, they are apt to contain hemorrhagic areas. Their growth may be quite slow or rapid and it is enhanced by anything that will give the tumor cells sufficient nourishment. Not infrequently a small tumor will, after an exploratory operation, grow to an enormous size because of the stimulating impetus that has been given to the tumor cells. This general principle holds true in all malignant neoplasms which frequently grow very rapidly after exploratory incision.

In order to demonstrate the glia fibers, it is quite important that autopsy be performed within eight hours after death as after this time it is very difficult. The tendency, however, of the tumor cells to radiate from the blood vessels and the gross appearance of the tumor are sufficient for an anatomical diagnosis. There is one characteristic feature of brain tumors that is observed at autopsy. Whenever the dura strips off in the manner that a banana peels, you may rest assured that there is a foreign growth beneath it, because these tumors force out the spinal fluid from the arachnoid space, leaving an apparently dry brain.

The following two cases emphasize the fact that these tumors may grow to a considerable size without showing any clinical symptoms.

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[1] Osler's Modern Medicine, Vol. V, p. 311.

* Presented at the Clinical Conference of the Neurological Department of the College of Physicians and Surgeons July 8, 1918.

CASE I

The first case, T. B., was admitted to the Manhattan State Hospital November 17, 1917, and terminated fatally December 17, 1917. He was a Greek forty-seven years of age. All that we know of his history is that he was admitted to the Metropolitan Hospital, October 12, 1917, having been discharged from the workhouse. Physical examination at that time showed delayed tactile sense about the abdomen and an old scar on the genitals. He was markedly depressed and filthy in habits. A diagnosis of cerebrospinal syphilis was made and he was transferred to Bellevue Hospital on November 14, 1917. Here he manifested dullness and stupidity and his uncleanly habits continued; he was very resistive to care, noisy and irritable. His memory was very faulty for both remote and recent events, he was disoriented as to time and place, was dull of comprehension, had a poor understanding of his situation and no insight what ever into his condition. His replies to questions were very irrelevant. Examination showed pupils unequal and immobile; contraction of left arm, patellar reflexes hyperactive. There was incontinence of urine and feces and evidence of arterial hardening.

Physical Examination.—This was made at the Manhattan State Hospital and showed some anemia and arteriosclerosis; blood pressure, 145/95; pupils unequal, irregular and sluggish, almost stationary; increased knee jerks; contracture of left arm; drawing of mouth to the right; and fine tremor of the tongue and fingers. Lumbar puncture on November 27 showed negative globulin, no cells and a negative Wassermann. Mentally, he was simple, stupid, demented and uncleanly, and rambling in his statements. He was somewhat elated, had no hallucinations or delusions, knew he was in a hospital but could not give the date of his admission, failed to recall any of the retention tests after five minutes, and his grasp on recent and remote events was equally impaired. He stated that he had had two chancres, one ten and one seven years ago.

On November 23 another examination was made. At this time he was bedridden and could not walk or stand without assistance. There appeared to be a flattening of the left side of the face and also some contracture of the left arm and leg. Pupils were widely dilated and there was no reaction to light or accommodation. He held himself quite rigid, but the patellar reflexes were active. It was impossible to make any sensory tests owing to lack of coöperation on the part of the patient. A diagnosis of psychosis was made, but it was left in the unclassified group owing to lack of data. He gradually became weaker and went into coma in which he died.

Autopsy.—This was performed twenty-six hours after death. The body was that of a well-developed and well-built man. The left arm was flexed and the left thigh was somewhat adducted and inwardly rotated. The left extremities appeared more atrophied than the right.

Head.—Calvarium stripped readily leaving a very pale yellow looking dura beneath it. The dura was incised and stripped from the brain like a banana peel, leaving a very dry surface beneath it.

Brain weighed 1,560 grams. The entire pia had an unusual dry, pale yellow appearance. The vessels were not injected and there was a definite bulging in the right tempero-parietal region. This bulging had a definite increased consistency, and its margin gradually faded into the surrounding tissue. Horizontal section of the brain revealed a hemorrhagic infiltrating tumor, ten by twelve centimeters, which pressed upon the right lateral ventricle, especially on its posterior horn; it also pressed upon the right internal capsule and encroached upon the left internal capsule by pressing on the lateral ventricle, Fig. 1., p. 311. Hemorrhagic areas were seen.

Cord.—This showed nothing macroscopically.

Microscopic Examination. *Brain.*—Sections from the left first frontal, left first and second temporal and left paracentral convolutions showed a moderately thickened pia the thickening being essentially due to fibrous tissue in which there was considerable edema. The vessels of the pia were not thickened. All sections showed a normal cell arrangement. The vessels in the sections showed no perivascular exudation or arteriosclerotic changes.

Section from the tumor showed typical glia cells with hemorrhagic areas between. The tumor consisted of loosely arranged rather small cells with the protoplasmic processes interlacing in a network, Figs. 2 and 3, p. 312. There was considerable fiber formation.

Cord.—Sections from the cord, taken at various levels, showed a collection of cells about the central canal of the same structure as the cells of the tumor, Fig. 4, p. 312. This collection of cells is nothing more than a hypertrophied ependymal layer lining the central canal. There was no column degeneration seen in the Weigert and Marchi preparations.

Optic Nerves.—There was distinct atrophy in both discs.

Thoracic and Abdominal Organs.—These showed nothing of significance, except an interstitial fibrosis of the spleen.

ANATOMICAL DIAGNOSIS.—Glioma in right temperoparietal region, showing hemorrhages; bilateral optic atrophy.

CONCLUSIONS.—Here was a man about forty-seven years of age who, as far as can be learned, only began to show symptoms three months before his death of a condition that must have existed for years, and then the symptoms were easily attributable to cerebrospinal syphilis, a diagnosis which was made at one of the hospitals to which he was taken. Later he showed considerable dementia, faulty memory and disorientation, unequal and immobile pupils, contracture of left arm, very active knee jerks and incontinence of urine and feces. Still later, in fact a month before his death, he showed unequal, irregular and sluggish pupils, left hemiplegia, facial palsy, incontinence of urine and feces and mental degeneration. The serological findings in the spinal fluid were absolutely negative, but in the scars on the genitalia, and according to the patient's own statement, there was evidence of a luetic infection. Nevertheless, on postmortem examination a highly cellular glioma was found in the righ temperparietal region.

CASE II

The second case, W. C., was admitted to the Manhattan State Hospital, from Bellevue Hospital, on March 23, 1918, and died on April 15, 1918. He was an American, aged sixty-four years. The report from Bellevue stated that he had been brought there, from the Central and Neurological Hospital, with maniacal tendencies, constantly undressing himself and attempting to jump from the window; he was in rather poor physical condition, with a left hemiplegia; pupils responded to light, but there was incontinence of urine and feces. In addition he was suffering from arteriosclerosis. He showed impaired judgmet and faulty memory, was disoriented and had vague ideas of a persecutory nature.

PHYSICAL EXAMINATION.—This was made at the Manhattan State Hospital and showed a man in poor physical condition, with arteriosclerosis, advanced arcus senilis, systolic blood pressure of 170, left hemiplegia, pupils with somewhat diminished reaction to light and accommodation and limited in range and contracted, tongue deviating to right when protruded, face drawn to right, and slight contractures at left elbow and left knee joint. There was incontinence of urine and feces. Wassermann test of the spinal fluid was negative, but there were twelve cells and positive globulin. Mentally, he was silent, irrelevant, incoherent and somewhat depressed; disoriented for place and person; some date discrepancies; tendency to fabricate; very little school knowledge; fair insight and judgment. There was a slight tendency to perseveration; he was rather dull and it was somewhat difficult to gain his attention. As in the previous case, the patient's lack of coöperation made it impossible to conduct a thorough neurological examination with any satisfactory results. A diagnosis of psychosis with cerebral arteriosclerosis was made and therapy was adjusted to ameliorate his condition, but he grew weaker and finally died nearly a month after his admission.

AUTOPSY. This was performed twelve hours after death. The body was that of a fairly well-developed man showing considerable emaciation with marked loss of subcutaneous tissue.

Head.—Calvarium stripped easily and left a clear dura beneath it. On incision of the dura a moderate amount of cerebrospinal fluid escaped. There was no pachymeningitis.

Brain weighed 1,405 grams. It was of moderate size and did not appear asymmetrical. After it was fixed in formalin, there was definite reduction in size of the right hemisphere, there being an easily demonstrable loss of substance over the parietal region on the right side. The pia was moderately thickened over the frontal and parietal areas and showed a somewhat milky exudate in the same region. The base of the brain over the gyri recti and interpeduncular spaces showed a somewhat thickened pia. The vessels were collapsed. The frontal lobes showed moderate cerebral atrophy and the convolutions were separated by deep fissures. Horizontal sections of the brain showed an area in the right parietal lobe, five by six centimeters in size, containing many hemorrhagic areas. It occupied the subcortical region of the superior and inferior parietal lobules, the supramarginal gyrus, the posterior central gyrus, and the posterior third of the superior temporal gyrus, and was limited internally by the posterior part of the claustrum and anterior portion of the right optic radiation. This area definitely pressed upon the internal capsules on the right side and upon the right optic radiation, Figs. 5, 6 and 7, pp. 313-315. There was considerable edema of the brain tissue immediately surrounding the new growth.

Spine.—The spinal cord was removed and showed nothing remarkable on gross section.

Thorax.—The thorax was flat; sternum stripped easily.

Heart showed nothing of significance.

Aorta, throughout its entire course, showed definite areas of bluish gray patches with definite atheromatous areas in its abdominal portion.

Lungs showed left obliterative pleurisy and bilateral bronchopneumonia.

Abdomen.—Abdominal organs showed moderate visceral arteriosclerosis and diffuse nephritis.

Microscopic Examination. *Brain.*—The pia is thickened and edematous, the thickness being due to connective tissue. The vessels of the pia show definite arteriosclerotic changes. The frontal and straight gyri sections show definitely thickened vessels and retracted tissue about the vessels. There is a decided loss of nerve cells in the second and third layers of the cortex. There is moderate satellitosis. Some of the nerve cells show distinct loss of granules and the nuclei are absent in others, while many nerve cells appear swollen.

Tumor.—The sections from the tumor show a growth consisting of neuroglia cells which are arranged in a loose meshwork around the blood vessels, Figs. 8 and 9, p. 316. Many mitotic figures are present.

Cord.—Weigert and Marchi preparations of sections from the cord reveal slight degeneration in the left crossed pyramidal tract and, to a lesser degree, in the right uncrossed pyramidal tract.

Aorta.—The intima shows excessive fat tissue with irregularly thickened patches in the intima. The vessels of the adventitia are free from any perivascular exudate.

Lungs.—Sections show definite congestion of the alveolæ and alveolar walls with many polymorphonuclear leucocytes and red blood cells and a good many bacterial clumps. No fibrin was seen.

Kidneys.—Capsule is thickened. From the capsule, strands of connective tissue run into the cortex. The glomeruli show congestion; many of the capsular spaces are filled with débris. Some glomeruli are replaced by connective tissue. The vessels are thickened and show splitting of the internal elastic membrane of the larger vessels, with definitely hypertrophied intima. The tubules are filled with débris, and their cells show various changes from simple cloudy swelling to complete destruction of the cells.

Anatomical Diagnosis.—Cerebral arteriosclerosis; glioma of right parietal lobe; moderate cerebral atrophy; left obliterative pleurisy and bronchopneumonia bilaterally; aortitis; moderate visceral arteriosclerosis; and diffuse chronic nephritis.

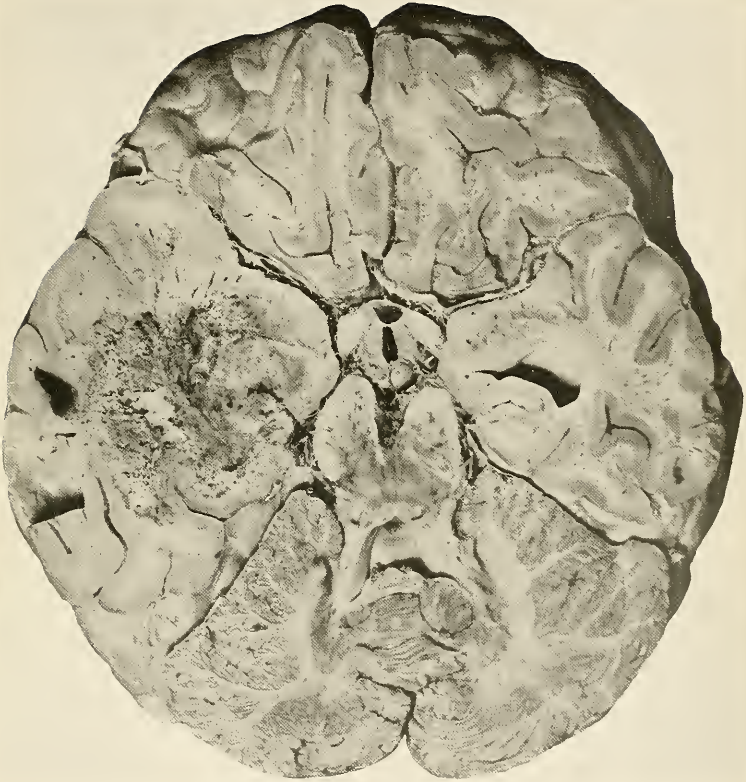

FIG. 1. HORIZONTAL SECTION SHOWING EXTENT OF TUMOR

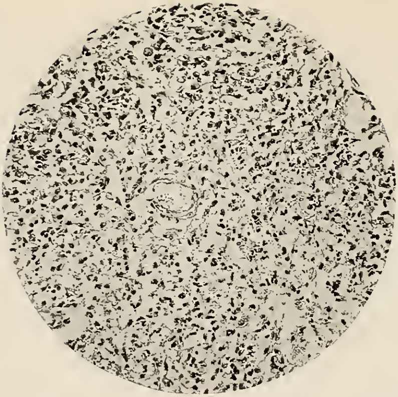

Fig. 2. Showing Type of Tumor Cell
(x 200)

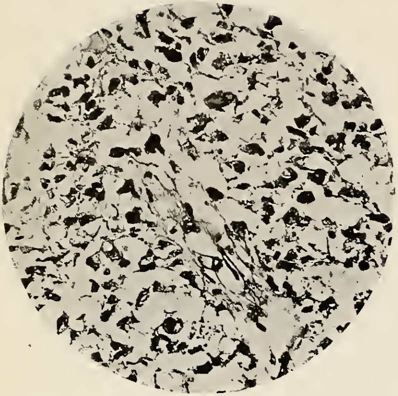

Fig. 3. Showing Type of Tumor Cell
(x 400)

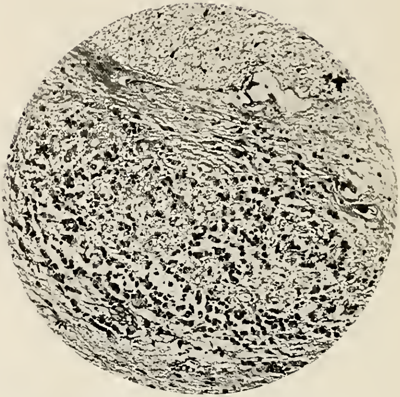

Fig. 4. Showing Resemblance between Cells of Tumor and Ependyma (x 200)

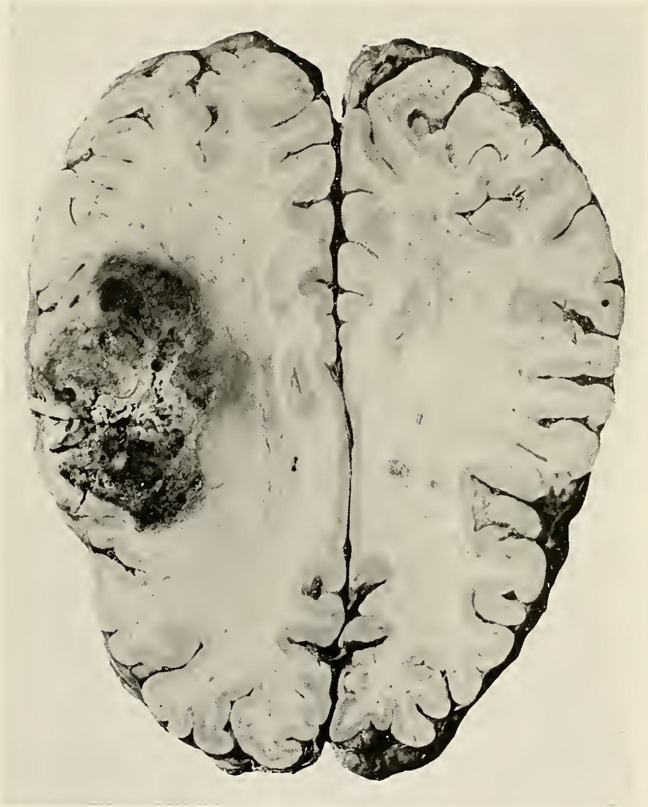

Fig. 5. Showing Extent of Tumor in its Various Planes

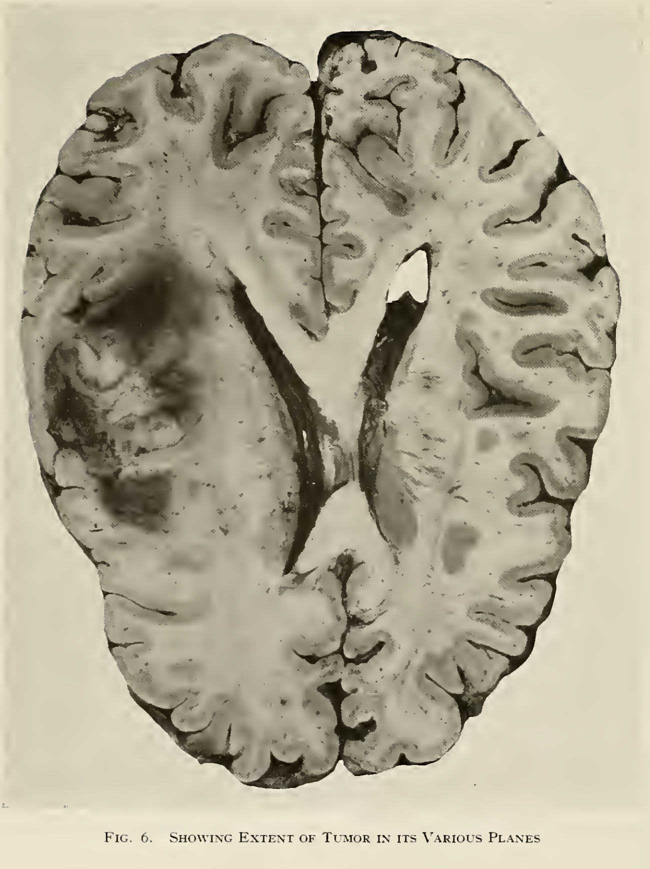

Fig. 6. Showing Extent of Tumor in its Various Planes

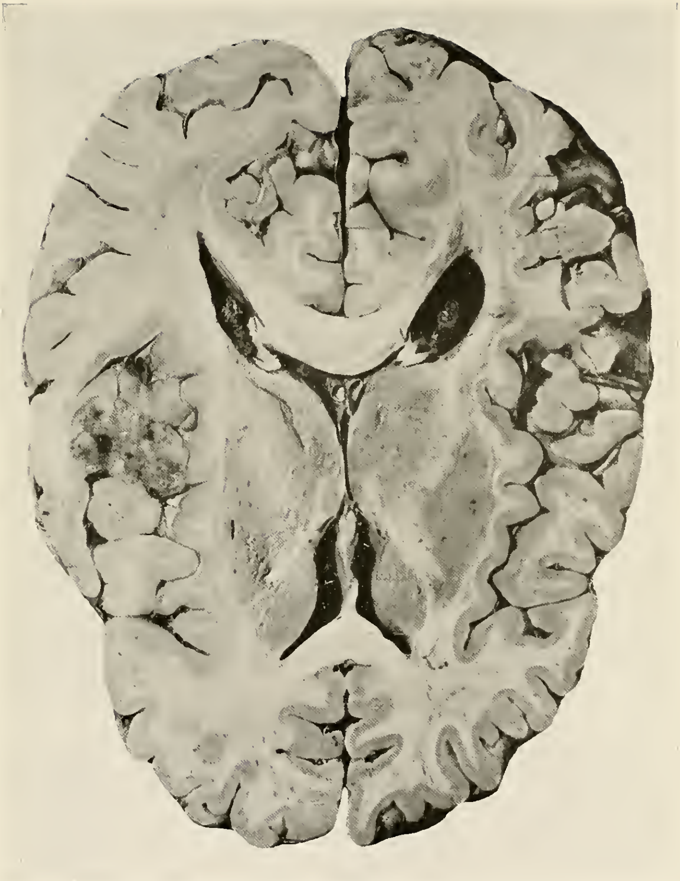

Fig. 7. Showing Extent of Tumor in its Various Planes

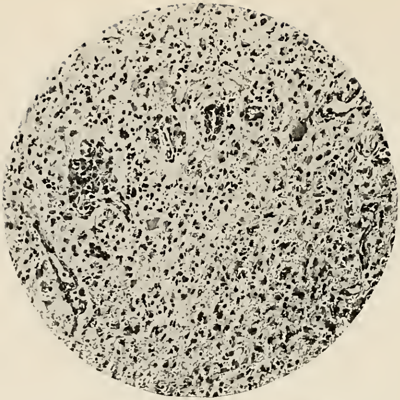

Fig. 8. Showing Type of Tumor Cells (x 200)

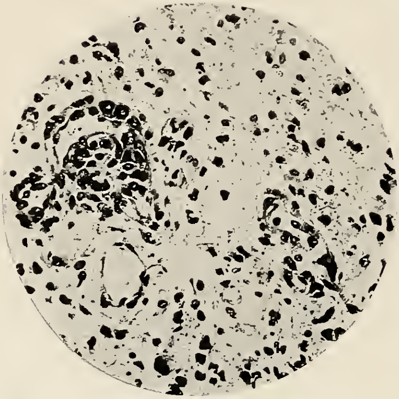

Fig. 9. Showing Type of Tumor Cells (x 400)

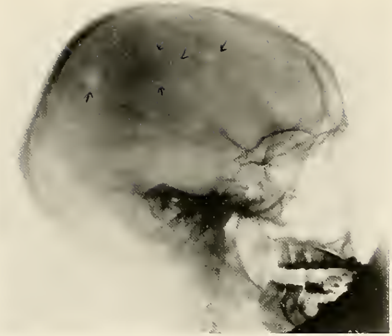

FIG. 1. CARCINOMA OF SKULL IN FIRST STAGES
Note somewhat irregular outline of defects

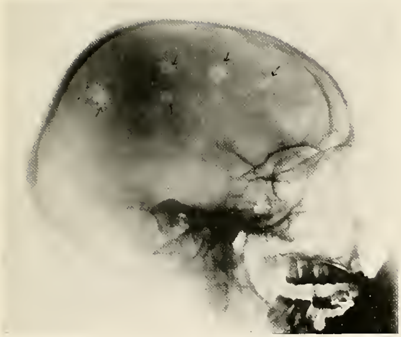

FIG. 2. CARCINOMA OF SKULL IN MORE ADVANCED STAGE
Same case as Fig. 1, but examined fourteen days later. Note increase in size of bone defects since previous examination

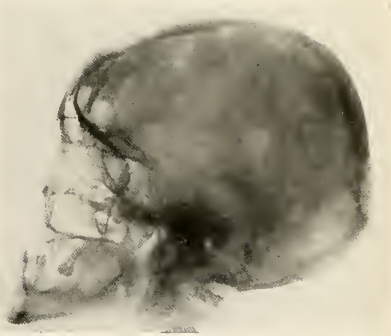

FIG. 3. CARCINOMA AFTER THYROID CANCER

[*Illustrations for "Metastatic Carcinoma of the Skull, by Thomas Scholz," M.D., page 319*]

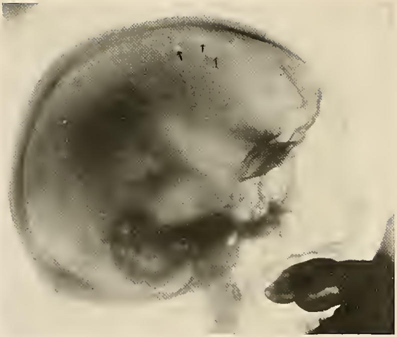

Fig. 4. Impressions by Enlarged Pacchionian Bodies
Note sharp, well-defined outline of bone defects

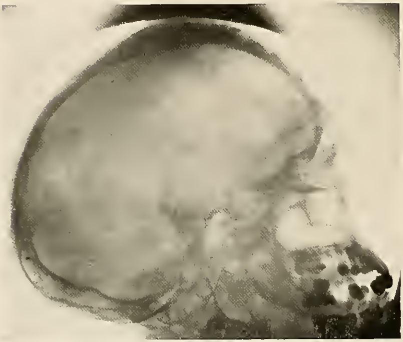

Fig. 5. Involvement of Tabulae

[Illustrations for "Metastatic Carcinoma of the Skull," by Thomas Scholz, M.D., page 319]

CONCLUSIONS.—In this case, we have a man aged sixty-four who was treated for hemiplegia and loss of sphincteric control for about one year prior to his death; who showed general arteriosclerosis, blood pressure, systolic, of 170, sluggishly reacting pupils; positive globulin and twelve cells, but negative Wassermann, in the spinal fluid; mentally somewhat incoherent and rambling; patchy amnesia and preservation of personality; who, on postmortem examination showed a glioma in the right parietal region, cerebral and visceral arteriosclerosis and bronchopneumonia.

SUMMARY

The first case shows (1) that a tumor may by its growth drive the small amount of fluid out of the arachnoid space so that when the dura is removed the brain is found in a dry condition; (2) the enormous size to which a tumor may grow; (3) that such a condition may exist in the brain for years without any clinical manifestations; and (4) that the real condition would not have been detected if autopsy had not been performed, showing the value of postmortem examination if it be for nothing more than for proper diagnosis.

The second case shows (1) the highly vascular nature of gliomata; (2) the tendency of the tumor cells to grow around blood vessels; and (3) clinical signs, chiefly of cerebral arteriosclerosis and apparently not at all referable to the brain tumor.[2]

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[2] I am indebted to Professor Francis Carter Wood for the microphotographs, and to Dr. M. B. Heyman for allowing me to present and publish these cases.

METASTATIC CARCINOMA OF THE SKULL

ITS ROENTGENOLOGICAL DIAGNOSIS AND ITS FREQUENCY

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It is a well-established fact that carcinoma of the bone is always secondary, never primary. This is of extreme importance and interest to both clinician and roentgenologist. Wherever, therefore, we find radiographic evidence of bone carcinoma, we know definitely that somewhere in that patient's organism there is a primary carcinomatous focus, whether or not that focus has been discovered by the clinician. Such positive x-ray findings, therefore, should always act as a stimulus to the clinician to improve his diagnostic methods in certain cases of malignancy; and to the pathologist, in his autopsy findings, never to overlook—as has often been done—a small carcinomatous focus.

The importance of this diagnostic aid in certain obscure cases, which later on turned out to be malignant, was but gradually realized, both by the clinician and by the roentgenologist himself. It is no reflection upon the diagnostic acumen of the average clinician that he has only recently familiarized himself with the fact that a patient may have a very small primary carcinomatous focus somewhere within the body (in the thyroid, for instance) unrecognized and without any local symptoms or signs, but with extensive carcinomatous involvement of the osseous system; that an area of tenderness on the cranium with, or perhaps without, local swelling, and associated with an indefinite history of cephalalgia, may be the first sign in even as remote an organ, for example, as the prostate; or that what appears to be a simple sciatica, especially if bilateral, may be due to carcinoma of the body of the fourth or fifth lumbar vertebra, secondary to a small carcinomatous nodule in the thyroid.

The roentgenologist, on the other hand, failed for a long time to realize and, alas, is still somewhat somnolent in awakening to the great diagnostic possibilities that lie open to recognition by his tube and his personal interpretation if he would but make full use of his opportunities—if he would not only take 'pictures' according to the requisition slip and interpret them, but if he would also try to get some understanding of the clinical side, in those so frequent obscure cases, and then arrange the procedure of the x-ray examination accordingly. Let me illustrate. In hypernephroma it is rarely possible to make the roentgenological diagnosis until late in the pathological process. Owing to the frequency of osseous metastasis in hypernephroma, examination of the skeletal system not uncommonly reveals malignant foci: thus, with these data and the clinical symptoms suggestive of renal disease, one may draw positive conclusions. Herein lies the value of a more general examination based upon experience. The roentgenologist only too often forgets that in the fluoroscope he has a powerful and extremely convenient method of getting quick information in regard to gross pathological changes in the respiratory and gastro-intestinal tract and in the osseous system with the exception of the spine, pelvis and ribs. He not infrequently might be able to clear up, in a quick and most brilliant manner, some persistently obscure case—for instance, by finding a metastatic bone involvement.

When looking for possible metastatic carcinoma in the osseous system we were in the habit of concentrating our attention mainly upon the upper parts of humeri and femora and the lumbar spine. The other parts of the skeleton were very often overlooked. While studying normal lungs and gastro-intestinal tracts at the Mt. Sinai Hospital by means of the fluoroscope I found, accidentally, quite a number of various, more or less advanced, bone lesions, several of which finally proved to be of a carcinomatous nature. Later on I paid more attention during my fluoroscopic and radiographic studies to the skeleton. In regard to the bone carcinomas, discovered either fluoroscopically or radiographically, I was struck by the relatively great frequency of involvement of the skull. Out of twenty-four cases diagnosed as bone carcinoma in the years 1911–1914 at the x-ray department of the Mt. Sinai Hospital, there were eight cases of skull metastases. Later on, in the Montefiore Hospital with its rich cancer material, I continued paying special attention to skull metastases. Gradually a certain routine procedure was worked out according to which in every cancer case—indeed in every case where there was the slightest suspicion of malignancy, and in all other obscure cases, either before or after the patient's death—the following bony parts were x-rayed: skull; thorax with shoulders; lumbar spine; pelvis; and hips. Here again I was greatly surprised at the frequency of skull metastases. Out of one hundred and twenty-two cases diagnosed as metastatic bone carcinoma, fifty-four showed involvement of the skull, either alone or in combination with other bony parts, indicating that the skull, among the various parts of the skeleton, is by far the most frequent place for metastases. The next most frequent seat of involvement was found to be the upper portion of the femur; namely, in twenty-nine cases. With the exception of two, all the cases of carcinoma of the skull were of the osteoclastic type. In the following pages, when speaking of skull carcinoma, we will always have this latter type in mind. Osteoplastic carcinoma, frequently occurring in the spine, and especially secondary to carcinoma of the prostate, is so extremely rare in the skull that we may leave it out of consideration altogether.

Is the great frequency of metastatic carcinoma of the skull really so surprising; as a matter of fact should we not rather expect it? A brief consideration of the most plausible theory in regard to formation of a metastatic carcinoma focus in bones gives us some enlightenment. According to Recklinghausen the arrangement of the venous canals in bones *per se* presents favorable conditions for carcinomatous implantation; especially if those canals are very numerous, sometimes even forming an entire network of venous vessels. We think immediately of the skull and bodies of the vertebrae in which more than in any other parts of the skeleton, the veins are comparatively very large; their walls only slightly collapsible; and the blood stream, in those venous canals, very slow along the wall of the vessel, though slightly more rapid in the center. Any foreign body carried in the blood stream of these veins is being pushed towards the wall of the vessel. Thus opportunity is afforded for the foreign body—in our instance the carcinomatous cell—at a point where there is a slight roughening of the vessel wall, to become deposited and start carcinomatous vegetations which latter gradually grow at the expense of the surrounding bone substance causing what radiographically shows up as a bone defect. This one point of Recklinghausen's theory on cancer metastases in bones should call attention to the probability that the skull may constitute one of the seats of predilection for metastatic carcinoma. To dwell upon the other factors, like static moments, etc., aiding in producing such pathological conditions would lead too far.

Are we able to diagnose roentgenologically metastic carcinoma of the skull? And if so, at what stage?

Minute carcinomatous foci, in the skull as well as in any other bony part, cannot be demonstrated. In other words *one* negative radiographic finding does *not* exclude the presence of bone carcinoma at that particular time. A complete reëxamination should always be made after some time, because minute foci, once present, increase in size quite rapidly. Demonstrable osteoclastic carcinoma of the skull, as of all other bones, manifests itself on the x-ray plate at first as a small, somewhat round bone defect, gradually and steadily increasing in size as shown by each reëxamination, Figs. 1 and 2, p. 317. There is not the slightest evidence of bone condensation or bone sclerosis. The outline of the defect is never sharp or clean cut, but rather ragged in appearance. The gradual enlargement is very characteristic. It is evidently a continuous process, new small foci showing at each reëxamination and again growing rapidly. In no other part of the skeleton is there such a rapid formation of new foci. The development of this entire process can be watched and demonstrated radiographically in the most beautiful manner. Some of our patients were followed up radiographically from the first demonstrable small focus in the skull up to the time when the entire skull appeared studded with small and large bone defects, Fig. 3. p. 317.

In differential diagnosis one must be careful not to interpret the Pacchionian depressions (*foveolae granulares* Pacchioni), caused by abnormally large Pacchionian bodies, as beginning metastatic carcinomatous development of the skull. The former, in some elderly individuals, appear as small round defects in the skull bones. These depressions are, however, sharp in outline and usually situated in the upper portions of the parietal region, Fig. 4, p. 318. If there is doubt, the diagnosis can be settled by reëxamining the patient about two weeks later, when, if the defects are of the same size, it is safe to say that they are not due to carcinoma.

Syphilitic lesions of the skull, primary sarcoma, or any other bone lesions, will always show, besides the process of bone destruction, more or less marked periosteal reaction, new bone formation, bone condensation, etc. There is, however, one bone lesion; rare in its occurrence, which once led me astray, namely, bone changes in Hodgkin's disease. In that particular case, which will be published more exhaustively later, there were quite a large number of bone defects of varying size in the skull, Fig. 5, p. 318. The appearance of the defects resembled closely those of carcinoma. As the roentgenological examination of the lung seemed to indicate an invasion of the lung substance by the mediastinal tumor masses, the diagnosis of malignant lung tumor with metastases to the skull was made. Autopsy showed a case of typical Hodgkin's disease, the defects in the skull being due to exuberant lymphoid tissue.

Good clean-cut plates, showing the bone structure in minute detail, are, for a correct roentgenological diagnosis in cases of metastatic carcinoma of the skull, a *conditio sine qua non*. In no other pathological condition of the osseous system is a poor, or even an average plate, so misleading as in carcinoma.

Conclusions: Metastatic carcinomatous involvement of the skull is extremely frequent. It is found sometimes accidentally. Its x-ray diagnosis is comparatively easy. Clinically it can be diagnosed, as a rule, only in its last stages. The clinician, as well as the roentgenologist, should bear in mind that radiographic examination of the skull may be of great help in the diagnosis of an obscure case or in cases where the clinical evidence only slightly points in the direction of malignancy.

ANEURYSM OF THE INTERNAL CAROTID INVOLVING THE ABDUCENS NERVE *

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Aneurysms of the cerebral vessels are not uncommon, especially as noted by men of large postmortem experience, but they are sufficiently rare in clinical observation to warrant the reporting of those that are diagnosed during life. The following case is of unusual interest, not only because it would at first lead one to suspect a purely vascular type of luetic manifestion, but also because serological investigation reveals a problem of differential diagnosis between the meningeal or vascular origin of the patient's symptoms.

As our experience in serological diagnosis increases, we are impressed with the necessity of accurate study of the spinal fluid as well as the blood in all cases in which syphilis is suspected. Personally, I believe that the central nervous system may be involved for a period of years in many cases before giving rise to clinical signs of disturbance. Therefore, I feel that it is our duty to insist upon spinal fluid examination in every case that gives a positive Wassermann response in the blood. Certainly, one is not justified in finally classifying a case as a vascular type of lues without first having ruled out the existence of cerebrospinal involvement. Only by such procedure, in my opinion, can more extensive invasion of the nervous system be intelligently and successfully prevented.

The patient described below has now reached a stage where it is exceedingly doubtful as to the relief to be afforded by rigorous anti-specific treatment, but I present him chiefly to ascertain the opinion of the conference as to surgical, or other measures, with a view to relieving him of most distressing symptoms.

Patient: G. D.; Case No. 21986; male; aged 47; colored.

HISTORY. *Present Illness.*—The patient complains of double vision, severe headaches, and above everything else, an intermittent puffing in the right ear. He suffers also from vertigo of a vague nature, in which objects seem to be coming and going. He was always well up to the onset of these symptoms about eighteen months ago. The first in order of appearance was double vision, and, almost at the same time, a throbbing in his right ear was observed. The latter has varied considerably in intensity; some days he has scarcely noticed it. The double vision has been constantly present.

The patient began to suffer with severe headaches at the onset of his other complaints. They appear to have been occipital and biparietal chiefly. He describes them as a constant ache, with occasional darting pains. The horizontal position relieves the pain considerably.

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* Presented at the Clinical Conference of the Vanderbilt Clinic, July 22, 1918

The patient observes that he can immediately shut off the throbbing sensation by pressure on the right side of the neck. He states that it is similar to the sound of an exhaust pipe, especially when he is excited, or when he is intensely interested in something. Position does not affect the sound in any way.

The vertiginous sensations have not been constant. They have occurred about once a month, especially when the stomach has been empty. They are accompanied by nausea, but only rarely by vomiting. They have lasted about fifteen minutes. Often the patient has had to sit down in order to prevent his falling to the ground. He has no knowledge of the direction of rotation of objects. The somatic functions have been normal. Sleep is normal except during severe headaches.

Previous History.—It is interesting to note that the patient is one of seventeen children, all of whom died except two. Eight brothers and sisters have died in the past twenty-five years. None of them are said to have suffered from any nervous disease. The previous history and family history are otherwise negative. The patient denies venereal infection of any kind. His habits are good. He has several healthy children.

EXAMINATION

VOLUNTARY MOTOR SYSTEM. *Abnormal Attitudes and Deformities.*—None are noted.

Gait.—Normal.

Coördination.—Normal for all equilibratory and non-equilibratory tests.

Skilled Acts.—Normally performed.

Reflexes.—*(a) Deep.* These are all present and equal right and left. They are not increased.

(b) Superficial. These are all present, right and left, but there is a questionable increase in response of the left lower lateral abdominal and left cremasteric as compared with the right. Plantar flexion is normal.

Abnormal Involuntary Movements.—None can be detected.

Muscle Strength.—The patient is of a vigorous and powerful physique. There is no weakness in any of the muscle groups.

Muscle Status.—There is no diminution or increase in volume, tone or consistency. Electrical reactions are not tried.

Abnormal Associated Movements.—These are not tested.

NERVE STATUS. — Nothing abnormal is noted.

GENERAL SENSORY.—Touch and pain only are tested and no abnormality is found in any part of the body.

CRANIAL NERVES.—*Olfactory and Nose.*—Not examined.

Optic and Oculomotor Apparatus.—Dr. M. J. Schoenberg of the Ophthalmological Department, who kindly referred the patient to me finds no abnormality whatsoever, except a paresis of the right external rectus. My findings agree with his in every respect. There are no pupillary abnormalities. The fundi are negative.

Acoustic and Ear.—Careful examination reveals no abnormality. There is no middle or inner ear disease. With the stethoscope placed directly against the skull, immediately in front of the right ear at its upper part—especially after vigorous exercise—a soft, systolic, seemingly far-distant whiff can be heard. The patient, upon request, can stop it by moderate pressure upon the vessels in the neck, and it recurs upon relieving the pressure. It can also be stopped by the patient when the head is turned to the extreme right. It disappears at times, regardless of the position or activity of the patient. On one occasion, after violent exertion, the sound could not be detected. The sound cannot be heard in any other area than the one indicated.

Trigeminus and Mouth.—The jaw movements are normal.

Facial and Face.—Normal.

Glossopharyngeus, Vagus and Larynx.—Nothing abnormal can be found.

Hypoglossus and Tongue. — The tongue movements are normal.

MENTAL STATUS.—This is carefully noted. The patient is of unusual intelligence, normal in his reactions, and not unduly depressed. There are no delusions, exaltation or other abnormalities.

SYSTEMIC.—*Tegumentary, Glandular, Cardiovascular, Respiratory, Gastro-intestinal, Genito-urinary, Skeletal.*—These have been thoroughly investigated and nothing abnormal has been detected excepting a systolic pressure of 178 mm.

LABORATORY TESTS. *Wassermann.* — The blood was four plus.

Lumbar Puncture.—The spinal fluid was four plus to 0.2; 2 cells; plus globulin; and Lange's gold colloidal test was 544433100—representing a paretic curve.

Urine.—(Taken at a later date after an intravenous salvarsan.) This revealed a slight trace of albumen, many casts and a few white blood cells. The specific gravity was 1015.

X-Rays.—Those of the skull have been negative.

DIAGNOSIS

The patient is suffering from the results of a combined vascular and neural lesion. One naturally asks first whether the intracranial murmur might be due to some meningeal inflammatory process or gumma involving the internal carotid and its vulnerable neighbor, the abduceus, secondarily. In reply to this I would say that the patient presents no clinical signs of cerebrospinal lues eighteen months after the onset of his symptoms, although the positive fluid is direct evidence of invasion of the nervous system. He has been taking iodide of potassium in ten-drop doses for over a year, and it would seem likely that if the lesion were primarily meningeal the symptoms either would have diminished or increased in severity by this time.

The murmur, distinctly heard at times, is, of course, vascular in origin. The tests prove that the internal carotid or one of its branches is involved. I believe that syphilis has a predilection for cerebral vessels in the production of aneurysms. In endeavoring to localize the lesion one must bear in mind the fact that the sixth nerve is the most vulnerable of all the cranial nerves either by direct pressure, fluid changes, or increased intracranial pressure in general. Certainly the tumor is not of great size, or other cranial nerves or structures would manifest abnormalities. Therefore, one is tempted to hazard a guess that a small aneurysm of the internal carotid, as it passes along the cavernous sinus, exerts slight pressure upon its closest companion, the abduceus, at its outer side. But I think it highly improbable that one could be certain in this case. I recall that the main trunk is much more frequently affected than any of its terminal or communicating branches, excepting the middle cerebral. It is difficult to imagine a lesion of any of these affecting the abduceus indirectly without producing signs which we lack in this case.

Therefore, I believe that this patient's symptoms are due primarily to a vascular, luetic, degenerative process, involving simultaneously, at the onset, the sixth nerve.

The literature reveals several instances of intracranial aneurysms that were accompanied by headache, usually of a pulsating, continuous or paroxysmal type, such as that from which the patient suffers. Vertigo and distressing auditory sensations have been reported often, especially in internal carotid lesions. An audible murmur, curiously enough, is not frequently encountered.

The patient here presented is suffering acutely. I would like to ask the conference whether surgical measures, such as decompression, or ligation might afford relief. The prognosis from the standpoint of antispecific medication is bad. There have been cases of supposedly spontaneous disappearance of intracranial aneurysms, but, of course, many cases of rupture with hemorrhage are known to us.

OSTEITIS DEFORMANS WITH A REPORT OF TWO CASES

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The term osteitis deformans was first used by Czerny (1) in 1873, in describing a case of spontaneous curvature of the lower limbs. It is believed by Emerson (2) that this was a case of osteomalacia. In 1877 Paget (3) used this term for a group of cases which he described as a separate disease.

He described five cases in which he thought that the bone changes were the most important and remarkable. He stated that the disease began in middle life, was very slow in progress, might continue for many years without influence on the general health and give no further trouble than those which were due to the changes of shape, size and direction of the diseased bones. Even when the skull was hugely thickened he found that the mind remained unaffected. According to his description the long bones of the lower extremities and of the skull were most frequently and symmetrically affected. The bones enlarged, became softened and those bearing weight were unnaturally curved and misshapen. The spine sank and seemed to shorten with increased dorsal and lumbar curves, the pelvis might become wide and the neck of the femur almost horizontal. He further stated that in the earlier periods and sometimes through all its course the disease was attended by pains in the affected bones, the pains varying in severity and described as rheumatic, gouty, or neuralgic. There was no fever and no characteristic changes in the urine or feces. It was not associated with syphilis or any other disease except possibly with cancer.

Postmortem examination of the bones showed the results of an inflammation affecting the skull in the whole thickness; and the long bones in their walls principally, but also, in a characteristic manner, their articular surfaces. Paget (4), five years later, added further observations to his original description and stated that there was a uniformity in the general appearances, postures, and general movements of the patients which was unmistakable and from which alone a diagnosis could be made. The most characteristic changes, according to his later description, were the loss of height as shown by the low position of the hands when stooping; the low stooping with the round shoulders; the head far forward, and the chin raised as if to clear the upper edge of the sternum; the chest sunken toward the pelvis; the abdomen pendulous; the lower extremities curved; the ankles overhung by the legs and the toes turned out. The enlargement of the head, square looking or bossed, might add a further feature of distinctiveness, and also the slow awkward gait and the shallow costal breathing.

This disease is a rare one and in 1906 Clopton (6) was able to collect only seventy-five cases from the literature. In over thirty thousand cases at the Johns Hopkins Hospital, Hurwitz (7) found only three cases. In 1915, five cases were found by Da Costa (8) in over thirty-eight thousand admissions to the Jefferson Hospital. At the time of this report only two hundred and thirteen cases had been reported. A review of the literature since that time shows that eight additional cases have been reported.

In 1915 Da Costa, Funk, Bergeim and Hawk reported five cases, with complete metabolism studies in two cases, and also reviewed the literature. Their article is a classic and the studies were exhaustive in all fields. I shall quote some of their most important observations. They found from a review of the literature that the disease may occur at any age, cases having been reported in patients as young as eight years and as old as seventy-nine years at the time of onset, so that Paget's original observation that the disease occurs only in persons over forty years does not hold true.

Males are more frequently affected than females, both of my own cases being of the male sex.

Heredity has seemed to play a part in about seven per cent. of the cases reported. In one of the cases here reported the patient stated that his mother and brother were similarly affected, but was unable to give any detailed description.

Both of my patients showed negative Wassermann tests in the blood and spinal fluid. This finding has been confirmed in earlier studies by numerous investigators, and the administration of mercury and iodides over long periods has not shown any effect on the disease. Two cases have been reported, however, in which syphilitic changes in the osseous and arterial systems were found. The concensus of opinion in England and the United States is that syphilis is not a causative factor.

Dr. Aller G. Ellis made bacteriological examinations of excised portions of the tibia in two of the cases reported by Da Costa but his findings were negative.

There are no studies available to show definitely that this disease is of neurotrophic origin, although cases have been reported in which the central nervous system and peripheral nerves were affected. Thus Von Recklinghausen (9) found a chronic myelitis at autopsy. Gilles de la Tourette and Marenesco (10) found lesions in the medulla and the peripheral nerves which the former believed were due to senility. The condition has been described in a tabetic by Chartier and Descomps (11) and Mackey (12) described a case associated with Huntington's Chorea. One observer, Prince (13), believed that the condition was related to the myopathies of spinal origin. Klippel and Weil (14) reported a case which showed unilateral involvement of the skeleton accompanied by marked local hyperemia which suggested some nervous influence. The general opinion at present is that the disease is not dependent upon gross changes in the general nervous system.

Da Costa and his associates believe that the disease is a disorder of bone metabolism probably dependent upon the absence or perversion of the secretion of some of the endocrinous glands. An osseous disturbance is seen in cases of acromegaly and of gigantism associated with disease of the pituitary gland. Furthermore, osseous disturbances are seen in cretinism. According to Falta (15) there is a retardation of osseous growth; the bones are short and thick in cachexia thyreopriva while in morbus Basedowi the skeletal build is slender and at times the bones are weak and thin. According to Bircher (16) the thyroid gland influences bony growth even in the normal animal. Da Costa and his associates found calcification of the pineal gland in two of their cases. Tilney has shown recently that this gland is not a vestigial organ and the evidence at hand tends to show that its secretion influences in some way the early growth of the individual. It might be further added that the gonads certainly seem to have an influence upon osseous growth because in eunuchs the breadth of the shoulders and the pelvis shows a mid-form between a masculine and a feminine type and remains infantile, the bones, especially the long bones, remaining delicate while the sites of the mascular insertions are poorly developed. The closure of the epiphyses is delayed and ossification of the cranial sutures is also delayed. The head is small and flattened and the spinal column is especially short. The extremities are lengthened, particularly in their distal parts, thus causing a preponderance of the lower length over the upper, with a relatively large span width. In these conditions the sella is remarkably large but Falta does not believe that this indicates an increase in function in the pituitary gland. It would seem rather that there is an interrelation between the gonads and the other endocrinous glands which influence skeletal growth. No uniform lesions of the endocrinous glands have been described thus far in this disease. From the metabolism studies by Da Costa and his associates it was found that in the advanced case there was a pronounced retention of calcium, magnesium and phosphorus and a pronounced loss of sulphur. The findings were interpreted as indicating a stimulated osseous or osteoid formation accompanying the resorption of the highly sulphurized organic matrix. In advanced osteitis deformans the first step in the new formation of bone or osteoid tissue may be the production of a highly sulphurized matrix, transformed gradually by a calcification process which is accompanied by deposits of calcium, magnesium and phosphorus in this matrix. These writers supposed that in the course of this calcification process a certain amount of the sulphur of the matrix is replaced by the other elements mentioned, entailing the retention of calcium, magnesium and phosphorus with an increased elimination of sulphur. The metabolic picture is considered a reversal of that seen in osteomalacia.

CASE I

Patient: H. D.; male; fifty-eight years; married laborer; born in United States; admitted to Kings County Hospital, April 19, 1918, service of Dr. Benjamin F. Corwin.

HISTORY. *Present Illness.*—The patient complains of weakness in the legs when walking and states that this has been present during the past twelve years. For the past four years he has suffered with dyspnea on exertion.

Previous History.—He has never attended school and is unable to read or write. He has been employed as a laborer all his life and has been moderate in the use of alcohol and tobacco. He contracted gonorrhoea at eighteen years. He was married at the age of twenty-one years but his wife deserted him within a few weeks. He remarried at the age of thirty-five years. There were nine children and two miscarriages by this union.

Twelve years ago he fractured the left tibia at the knee and states that since then he has suffered with severe pains radiating from the knee to the ankle, first on one side and then on the other but chiefly in the bones of the legs. At times, however, he has had severe pains radiating from the shoulders to the wrists. He has never been confined to bed and there has never been any swelling in the joints. Since the fracture of the left tibia above mentioned there has been an ankylosis of the left knee.

In February, 1916, the patient fell, injuring the left side of his chest. At this time he noticed a swelling of the left leg. below the knee and dates the enlargement from this period. The swelling decreased after a few weeks, but since then the bones of both legs have appeared to enlarge. He claims to have had genu varum all his life, but for the past year the bowing of the left leg had increased noticeably. He has never noticed any enlargement of his head and has always worn a hat ranging in size from seven to seven and one-half inches. For the past four years he has worn a hat seven and one-half inches in size. During the past four years he has suffered with severe paroxysmal pains in the right axilla. During this time his ankles have been frequently swollen and he has also experienced dyspnea on exertion. As long as he can remember he has had nocturia, arising once or twice during the night. There is no history of polyuria nor polydipsia. About six months ago his wife remarked to him that he seemed to be shorter in stature. He states that his normal height is five feet, seven and one-half inches in his stocking feet. His present height in his barefeet is five feet, five inches. His weight has averaged from 190 pounds to two hundred pounds.

Family History.—The patient's father suffered with asthma and a brother has been alcoholic. There is no history of any disease similar to the one from which the patient now suffers.

VOLUNTARY MOTOR SYSTEM. *Attitudes.*—The patient presents a bent-over, ungainly appearance, is round-shouldered and the head projects forward, while the hands appear to hang much lower than normal. There is a marked scoliosis to the left in the thoracic region and a pronounced lordosis in the lumbar region. There appears to be a general sinking downward of the vertebral column in the pelvis. There is a marked forward bowing of both femurs and a well-marked forward and outward bowing of both bones of both legs. Apparently the bones of the upper extremities and of the feet are not affected. The head is enlarged and the temporal vessels are sclerosed. The enlargement of the head appears to be general but is especially marked in the right parietal region. The bones of the face are not affected. The left shoulder is considerably higher than the right and the clavicles appear enlarged. The pelvis appears abnormally broad and there is flaring of the iliac crests.

Gait.—The gait shows no abnormalities except that the patient presents an odd appearance because of the peculiar deformities present and there is some difficulty because of the ankylosis of the left knee.

Coördination. — (*a*) *Equilibratory.* — Shows no disturbance.

(*b*) *Non-Equilibratory.*—Shows no disturbance.

Skilled Acts.—Speech shows no abnormalities and there is no apraxia.

Reflexes.—(1 = Slight; 2 = Active):

(*a*) *Deep.*—	*Right*	*Left*
Jaw-jerk	1	1
Biceps	1	1
Triceps	1	1
Suprapatellar	1	0
Patellar	2	0
The left knee is ankylosed		
Achilles	2	1
Ankle clonus	0	0
(*b*) *Superficial.*—		
Upper lateral abdominal	1	1
Lower lateral abdominal	1	1
Cremasteric	1	1
Plantar flexion	0	0
Babinski	0	0
Schäfer	0	0
Gordon	0	0
Oppenheim	0	0
Chaddock	0	0

Abnormal Involuntary Movements. — Fine tremor of extended fingers. Twitchings; athetoses; spasms; convulsions—none.

Muscle Strength.—Muscle tonus is normal.

Abnormal Associated Movements. — None present.

NERVE STATUS.—No abnormalities.

GENERAL SENSORY.—*Touch, Pain, Temperature, Vibratory, Pressure, Muscle-tendon and Stereognosis.*—Normal.

CRANIAL NERVES AND HEAD.—*Olfactory and Nose.*—Normal.

Optic and Oculomotor Apparatus.—Normal.

Eye Grounds.—Normal.

Trigeminal and Mouth.—Normal.

Facial and Face.—Normal.

Acoustic and Ear.—Normal.

Glossopharyngeus, Vagus, Pharynx and Larynx.—Normal.

Spinal Accessory.—Normal.

Hypoglossus and Tongue.—Normal.

MENTAL STATUS.—Shows no psychotic manifestations and is of fair intelligence considering the lack of educational opportunities.

CRANIAL MORPHOLOGY.—See general description under abnormal attitudes and deformities.

Cranial Measurements:

Bitemporal	13.2 cm.
Biparietal	18 cm.
Fronto-occipital	20 cm.
Mento-occipital	21 cm.

SYSTEMIC EXAMINATION. *Tegumentary.*—No abnormalities.

Glandular.—Thyroid, thymus, tonsils, lymphatic glands—no abnormalities.

Cardiovascular.—(*a*) Pulse varies from 80 to 96 per minute, and shows some irregularity in force and frequency.

(*b*) *Blood Pressure.*—Systolic 145; diastolic 75.

(*c*) *Heart.*—Presystolic thrill at apex with accentuated first sound. Soft blowing systolic at ensiform cartilage, not transmitted. Heart enlarged to left.

Respiratory.—Chest is enlarged and emphysematous with moist râles at bases.

Gastro-intestinal.—No enlargement of liver or spleen but abdomen is short and wide and marked by a transverse furrow at level of pubis.

Genito-urinary.—No abnormalities.

Skeletel:

	Right	*Left*
Mid thigh	48.3 cm.	48.3 cm.
Mid calf	36.8 cm.	40.6 cm.
Mid arm	29.2 cm.	26.6 cm.
Right forearm	25.4 cm.	24 cm.
Right wrist	17.8 cm.	17.8 cm.
Right hand	22.9 cm.	22.9 cm.
Anterior superior spine to external malleolus	90.2 cm.	90.2 cm.

Pelvic Measurements:

Anterior superior spines	27 cm.
Crests	22 cm.
Anteroposterior	22 cm.

LABORATORY TESTS. (*a*) *Blood Count.*—Red blood cells 2,670,000; white blood cells 6,400; differential: polynuclears 66 per cent.; mononuclears 10 per cent.; lymphocytes 24 per cent.; hemoglobin 90 per cent.

Blood Wasserman.—Negative.

Spinal Fluid.—Cells 5; No globulin. Wassermann and colloidal gold test negative.

Urine.—Specific gravity 10.10 acid. No albumin, sugar or casts. Creatinine 1.9 per 100 cc. of blood. Urea 3.9 grams per 100 cc. of blood.

X-ray.—X-ray plates were taken of the head, hands, pelvis, femur, tibia and fibula. They revealed an increase in diameter in the long bones and changes showing the bone to be more or less spongy. The outlines are very ragged, especially in the right tibia which is much bent. The articular surfaces of the lower extremities are thickened and enlarged. There appears to be a loss of mineral substance in the bones. The skull is much thickened and appears to be very dense, so much so that the outline is not clear. The diploë are not seen.

CASE II

PATIENT: J. P.; male; sixty-three years; single; born in United States; admitted to Kings County Hospital, March 9, 1918, service of Dr. Benjamin F. Corwin.

HISTORY. *Present Illness.*—The patient complains of weakness in his legs and of severe generalized headaches for the past three years.

Previous History.—The patient has been employed as a sewing-machine adjuster for the past forty-five years. He received a common school education and was apparently an average student. He has been a user of alcohol to excess for a number of years. Venereal infection is denied. He has been deaf for the past twenty years.

Ten years ago he injured his left leg and since that time has been lame. As far back as he can remember he has had genu varum and he thinks that this condition became accentuated following the injury. His head has always been large and there has been no change as far as he can observe. He adds that all of the members of his family have had large heads and a nephew has always had to have his hats made to order.

For the past three years he has noticed a weakness in his legs and also a numbness over his ankles, knees, elbows and shoulders. In addition, he has suffered during this time with severe generalized headaches and vertigo, with lapses of memory for short periods of time. There is no history of convulsive seizures or fainting spells. On February 20, 1918, he failed to recognize his relatives and this condition lasted for several days during which time there was also loss of sphincter control. At the end of this time his mental condition again became normal and he has been well ever since. He does not know what his greatest height has been but his present height in his bare feet is sixty-four and three-quarter inches.

Family History.—The patient states that his mother and brother had a condition similar to the one which he now presents but is unable to supply any details.

VOLUNTARY MOTOR SYSTEM. *Attitudes.*—The patient walks with a peculiar waddling gait, swinging his arms forward and backward as he walks. He is bent forward and the shoulders are rounded. He presents a marked lordosis in the lumbar region. There is also a marked anterior bowing of both femurs and both bones of both legs. Both knees are enlarged and both tibiae appear greatly hypertrophied.

Gait.—The gait shows no abnormalities in itself.

Coördination.—(*a*) *Equilibratory*—shows no disturbance.

(*b*) *Non-equilibratory*—shows no disturbance.

Skilled Acts.—Speech shows no disturbance and there is no apraxia.

Reflexes.—(1 = Slight; 2 = Active):

	Right	*Left*
(*a*) *Deep:*		
Jaw-jerk	2	2
Biceps	2	2
Triceps	2	2
Suprapatellar	2	2
Patellar	2	2
Achilles	2	2
(*b*) *Superficial:*		
Supraumbilical	1	1
Upper lateral abdominal	1	1
Lower lateral abdominal	1	1

(*b*) *Superficial* (*Continued*)	*Right*	*Left*
Cremasteric	1	1
Plantar flexion	1	1
Babinski	0	0
Chaddock	0	0
Oppenheim	0	0
Gordon	0	0
Schäfer	0	0

Abnormal Involuntary Movements.—Twitchings; athetoses; spasms; convulsions—none.

Muscle Strength.—No paresis nor paralysis of any of the muscle groups.

Muscle Status.—Muscle tonus is normal.

Abnormal Associated Movements. — None present.

NERVE STATUS.—No abnormalities.

GENERAL SENSORY.—Touch, Pain, Temperature, Vibratory, Pressure, Muscle-tendon and Stereognosis—normal.

CRANIAL NERVES. *Olfactory and Nose.*—Normal.

Optic and Oculomotor Apparatus.—Normal.

Eye Grounds.—Normal.

Trigeminal and Mouth.—Normal.

Facial and Face.—Normal.

Acoustic and Ear.—Normal.

Glossopharyngeus, Vagus, Pharynx, Larynx.—Normal.

Spinal Accessory.—Normal.

Hypoglossus and Tongue.—Normal.

MENTAL STATUS.—Shows no psychotic manifestations and the patient appears to be of fair intelligence.

CRANIAL MORPHOLOGY.—The head is enlarged to a marked degree, especially in the parietal region. There is no enlargement of the bones of the face.

Cranial Measurements:

Biparietal	18 cm.
Bitemporal	14.5 cm.
Occipitofrontal	21 cm.

SYSTEMIC EXAMINATION. *Tegumentary.*—Normal.

Glandular.—Thyroid, thymus, tonsils, lymphatic glands—no abnormalities.

Cardiovascular.—(*a*) Pulse is of poor quality, irregular and varies from 64 to 84 a minute.

(*b*) *Blood Pressure.*—Systolic 190; diastolic 100.

(*c*) *Heart* is hypertrophied, the apex being in the sixth interspace 2 centimeters external to the nipple line. There is a soft blowing double murmur at the apex transmitted to the axilla. The second aortic is accentuated.

Respiratory. The chest is emphysematous with moist râles at bases.

Gastro-intestinal.—No enlargement of the liver or spleen. The abdomen is short and wide and marked by a transverse furrow at the level of the lower ribs.

Genito-urinary.—No abnormalities.

Skeletel.—The measurements of the extremities correspond on both sides. The pelvis appears abnormally broad and the crests are flaring.

Pelvic Measurements:

Interspinous	24.5 cm.
Intercrestal	31 cm.
Left oblique	24.5 cm.
Right oblique	24 cm.

LABORATORY TESTS. *Blood Count.*—Red blood cells 2,860,000; white blood cells 7,400; hemoglobin 90 per cent.; differential; polymorphonuclears 73 per cent.; small mononuclear 13 per cent.; large mononuclear 12 per cent.; transitionals 2 per cent.

Blood Wassermann.—Negative.

Spinal Fluid Wassermann.—Negative; no cells; globulin, and colloidal gold tests were negative.

Urine.—Specific gravity 1,015; acid. No albumin, sugar or casts.

X-ray.—X-ray plates were taken of the head, pelvis, hands, femur, tibia, and fibula. They revealed an increase in diameter in the right tibia while the left appeared normal. The right tibia also showed a very irregular outline with a marked bowing. The skull showed a marked thickening.

SUMMARY

We have then two well-defined cases of osteitis deformans or Paget's disease. They conform to the classical descriptions of this disease. In one of the cases, J. P., there is probably a similar condition in two other members of his family. Both of these patients claim to have had genu varum as far back as they can remember. In each of the cases there is a history of injury to the legs antedating the onset of the osteitis. I believe that in this disease we are dealing with a disturbance of the secretions of some of the endocrinous glands; but from the present status of the question one is unable to state definitely which, or how many of the glands are affected. The condition differs from acromegalia chiefly in that the bones of the face, hands and feet are not affected. Furthermore, the skull does not show the marked thickening in acromegalia that is so characteristic of the condition under discussion.

In conclusion, I desire to thank Dr. Benjamin F. Corwin, attending physician at the Kings County Hospital, for permission to study and report these cases, and also to thank Dr. Henry Wolfer for the roentgen ray pictures used with this article. Finally, I wish to express my appreciation for the valuable assistance rendered by Dr. Robillard of the House Staff of the Kings County Hospital.

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TUMOR OF THE HYPOPHYSIS*

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The case is presented as much for the purpose of establishing the diagnosis as for the opinion of the conference on the method of treatment. If my diagnosis is correct the question of surgical interference, considering the danger and difficulties, is a very important one.

Case No. 20285; A. C.; age forty-five; male; married; white; peddler.

History. *Present Illness.*—On January, 31, 1918, the patient was brought to the clinic complaining of blindness in both eyes and of spells of nervousness accompanied by unconsciousness. The onset of the condition was sudden and dates back about four years. In the summer of 1913, the patient began to have severe, constant, frontal headache, usually worse at night. He had no vomiting or dizziness at that time. This kept up for two years. In July, 1915, he suddenly fell unconscious, does not remember for how long. Was put to bed, where he remained a few days. The headaches persisted and even grew worse. In

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* Presented at the Clinical Conference, March 18, 1918

November, 1915, he again had an attack of unconsciousness, lasting a few minutes. The headaches becoming very severe, he took to bed and stayed there several months. In February, 1916, the patient went to sleep with perfect vision and woke up in the morning to find himself stone blind. The blindness lasted till May, 1916, when his vision suddenly returned. He could see perfectly well and read small print until August, 1917, when he again suddenly went blind and has remained so to this day. In the past two years the headaches have been less severe and frequently he is free from them. Ever since his first attack of unconsciousness, July, 1915, he has had similar attacks anywhere from one to six daily. They are always of the same character. He has a feeling as if something were rushing from the back to the front of the head, he becomes semiconscious, the head is jerked to the right, the whole body and all the limbs jerk constantly; sometimes he falls. The spell lasts a few minutes then slowly passes off. I saw him in one of these attacks, which the patient described as severe. In this instance, he knew it was coming, he had the jerky movements he spoke of, but was not unconscious, was perfectly aware of what was going on, but was thoroughly frightened and emotionally very much upset.

In the past month the patient has had no more attacks, due as he thinks to the medicine he got, which consisted of a little R. and S. and bromides. The patient states that on February 19, 1918, at about eight in the morning he suddenly saw everything in the room. To make sure that he had no hallucination he noted the arrangement of the things in the room. After two minutes he suddenly lost his sight, but his wife corroborates the accuracy of his description of the furniture of the room.

Aside from the above complaints there is little to record. The patient has normal appetite, is not fond of sweets, but drinks much water. His bowels are regular; sleep is undisturbed. Urinates two to three times at night and five times by day. His memory is good. He has been hard of hearing for a number of years. He thinks he gained some weight but does not know how much. Sexually the patient is normal.

Previous History.—He had mastoiditis in 1889 and typhoid in 1902. In 1911 he was operated upon for some nasal condition. In 1912 he was in bed for two weeks for what he thinks was some intestinal disturbance (obstruction?). Six months later he had a stone removed from the left kidney. Despite the last operation he still complained of pains for which he was given frequent injections of morphine. At one time he received injections of mercury which he thinks loosened his teeth for a while.

Family History.—Apparently this has no bearing on the case. The patient has two children, twins, who are deaf and dumb. Three other children are healthy. His wife had two miscarriages.

VOLUNTARY MOTOR SYSTEM.—Nothing abnormal was noted. The patient is a little unsteady, but he has no Romberg. There was some suspicion as to the accuracy of his F. to N. and H. to K. tests, but it is doubtful whether they are of any importance. The deep reflexes are a little lively, but equal, and the superficial reflexes are also present and equal, except possibly the left cremasteric. There is no clonus and no Babinski and there are no abnormal associated movements. No atrophies or hypertrophies and no loss of muscle strength. There is a coarse tremor of the fingers of both hands.

NERVE STATUS.—Normal.

GENERAL SENSORY.—Normal response to touch, pain, temperature all over the body. Muscle tendon and stereognostic senses are normal.

CRANIAL NERVES AND HEAD. *Smell.*—Impaired on both sides but probably not absent.

Vision.—Absent in both eyes; no perception of light. Fundus showed at the first examination a marked neuritis and a choking of the disk of about four diopters, also tortuous vessels and atrophy of the nerve. The pupils did not respond either to light or accommodation. They are irregular in outline, both are somewhat dilated, the right being smaller than the left. The mobility of the eye is normal, but there is a slow, coarse lateral nystagmus. Dr. Holden reports: "Recent postneuritic atrophy of each nerve. Discs still blurred and veins very tortuous. There is a papilledema of one diopter."

Trigeminus.—Normal motor and sensory. No corneal anesthesia. The teeth are somewhat separated but their occlusion is normal. Taste is normal.

Facial and Face, Glossopharyngeal (Reflex) and Hypoglossal and Tongue.—Normal.

Acoustic and Ear.—There is loss of bone and air conduction in the right ear and diminished conduction in the left.

Mental Status.—The patient is very emotional and cries easily. He is possibly a little dull mentally, and has difficulty in doing small sums.

Cranial Morphology.—Seems to be normal. Comparison with a photograph taken fifteen years before shows no noticeable differences.

Systemic.—The patient is well nourished, rather stout but he shows no special fat distribution. There is no abnormal distribution or absence of hair. The superficial glands are normal. Heart is normal. Blood pressure is 82 diastolic and 115 systolic. Respiratory apparatus normal. Genito-urinary.—Penis and testes normal.

Laboratory Tests.—Blood—Wassermann negative. Spinal Fluid—Wassermann negaative, cells 1, globulin plus, Lange negative. Urine showed neither sugar nor albumin, sp. gr. 1016. Sugar tolerance (100 gms. dextrose) is somewhat higher than normal. *X*-ray of sinuses shows them to be very large but clear. Three plates show a very large sella, the clinoid processes almost absent and the floor very much eroded.

Summary and Diagnosis.—The salient features of the case are: 1. A history of sudden onset of blindness lasting three months, with sudden complete recovery of vision which was normal for fifteen months, then again sudden blindness persisting to this day. 2. Severe headaches. 3. Hybrid epileptiform twitchings. 4. Doubtful attacks of unconsciousness. 5. Possibly polydipsia and poluria. 6. Choked disk, optic neuritis and postneuritic optic atrophy. 7. Impairment of smell. 8. Absence of abnormal physical findings, particularly no tegumentary or skeletal changes except possibly separation of teeth. 9. Negative serology of blood and spinal fluid. 10. Absence of sign of sinus involvement. 11. High sugar tolerance. 12. Enlarged and eroded sella turcica.

As the optic neuritis is the only abnormal somatic manifestation present, three or four possible conditions present themselves for diagnosis, namely: 1. Optic neuritis following infection of the sinuses. 2. Syphilitic optic neuritis. 3. Functional amblyopia. 4. Tumor of the pituitary giving choked disc and postneuritic atrophy.

The fact that the patient was operated upon for some nasal condition might lend plausibility to the first possibility, but the subsequent history and the *x*-ray of the sinuses, showing them to be clear, would seem to point against it. A toxic neuritis also seems to be excluded, and a distinct choking of the disc to about four diopters militates against both. Syphilis is less easily excluded. The history is not against the diagnosis. However, the negative blood and spinal Wassermann, while not positive, certainly are very strong presumptive evidence against syphilis. So, too, the absence of other eye-signs and involvement of ocular muscles and other somatic signs would point away from syphilis. Functional amblyopia is favored by the sudden onset and spectacular disappearance of the blindness, also by the hybrid twitchings and the emotional reaction of the patient, but the positive eye findings, I think, altogether exclude such a possibility.

There now remains the possibility of hypophysial tumor. The *x*-ray showing an enlarged and eroded sella turcica evidently points to a destructive growth of the pituitary. The history of the onset, it is true, is not characteristic, but it is not absolutely against the diagnosis. The severe headaches and choked disc, as well as the isolated optic nerve findings, speak for increased intracranial pressure. The impairment of smell and emotional changes may well be due to pressure on the olfactory nerves and frontal lobes. The increase in weight (?), the separation of the teeth, the polydipsia and polyuria and high sugar tolerance speak for hypophysial dystrophy. The sudden remission is difficult to explain, though a cystic condition of the hypophysis might do it.

Now as to the kind of pituitary dystrophy. We are accustomed to think of a hyperpituitarism as shown clinically in acromegaly and of a hypopituitarism in dystrophia adiposis genitalia. The findings in this case apparently point to neither, though the duration of the disease could have brought out a more marked clinical picture. That there is some hypophysial dystrophy may be inferred from the polyuria, polydipsia, high sugar tolerance, and probable gain in weight. Absence of acromegalic signs except separation of teeth, or absence of characteristic adiposis and hair changes, I think, need not deter us from making the diagnosis. It is possible that a cystic condition of the pituitary has destroyed just enough of the pars glandulosa and attacked sufficiently the pars nervosa to give the picture shown by the patient. For the present, therefore, it seems that we are forced to the conclusion that we are deal ing with a cystic tumor of the hypophysis, causing choked disc and postneuritic optic atrophy.

Assuming the diagnosis to be correct, the question of treatment looms up very large. Pituitary extract might be tried, of course; but it is doubtful whether any results will be attained. The next thing to be considered is surgical interference, particularly for possible restoration of vision. If the destruction of the optic nerve has not progressed too far, it is quite possible that removal of all or part of the tumor will relieve the pressure on the chiasm, or whatever part is being compressed, and restore some vision. On the other hand, the liability to recurrence would make the operation only palliative and not curative. Further, the great danger of the operation and the risk to life involved would make us hesitate to advise radical treatment. As the patient is not suffering very much just now and even his epileptiform twitchings have disappeared we would be justified in awaiting further development. The whole question, however, might be put before the patient and the burden of the decision allowed to rest on him.

DISCUSSION

Dr. Norman Sharpe.—As regards surgical interference, the chances are that all the damage has been done already, and we are once again too late. The patient has only slight headache, and the other symptoms seem to have subsided, so, whatever the lesion was—and it probably had a connection with the pituitary—it seems to have already subsided. If signs of increased intracranial pressure still persist—and this can easily be ascertained by measuring the pressure of the spinal fluid by lumbar puncture—then operation would be indicated. But if there is no increase in pressure, operation will not relieve the present outstanding feature—that of optic atrophy.

Dr. Sylvester Leahy.—I, also, feel that we have nothing to gain here by operative interference, particularly for restoration of sight, and in view of the fact that the patient has no active symptoms at the present time. I should, however, strongly recommend the further trial of therapeutic measures to see if further improvement cannot be gained. In view of the *x*-ray and the physical findings, there can be no doubt that Dr. Wechsler's diagnosis is the correct one.

Dr. Morris Grossman.—In view of the fact that there is a paralysis of the pupil, amblyopia tremor of the hands, nystagmus and some deafness, I believe this man has a corpora quadrigemina tumor. I do not see that there is anything to gain by surgical interference. The recurrence of the blindness is an unusual feature and may be due to involvement of the lateral geniculate body. It is possible, however, that the growth caused temporary pressure on the optic tracts and is now giving rise to choked disc through obstruction and not through increased intracranial pressure.

Dr. I. S. Wechsler.—Although I consider this to be a pituitary tumor, I think it is not a case for operation. Replying to Dr. Grossman, I doubt whether tumor of the corpora quadrigemina would give these symptoms. I presented this case principally to learn if others concurred in my diagnosis of hypophysial tumor and whether operation is at all advisable.

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A CONTRIBUTION TO THE STUDY OF TEMPERATURE REACTION IN NERVOUS DISEASES*

THE REACTION TO SIMULTANEOUS COLD AND HOT STIMULATION IN SYRINGOMYELIA, TABES DORSALIS, MULTIPLE SCLEROSIS, PERIPHERAL NEURITIS, AND A CASE PRESENTING A CAPSULO-THALAMIC SYNDROME

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OUTLINE

Introduction—Summary of the normal reaction to the double thermal stimulus
Consideration of individuals' reaction in the various phases of the test
Comparison of the syndromes studied
General tendencies observable in the reaction to double stimulation—tentative interpretation
Conclusions

The present study is a continuation of investigations of the reaction to a double thermal stimulus, preliminary results of which were published in the NEUROLOGICAL BULLETIN for March, 1918. The former paper dealt with the reaction of normal subjects. The present paper is a study of twenty-two patients suffering from organic nervous diseases.

The general method of the experiment was to provide several observations in each of which the reaction-time was measured for a series of stimuli combining simultaneous and separate cold and hot stimulation. For details of the method, description of the apparatus, plan of the series, etc., the reader is referred to the previous article.[1] The patients included six cases of syringomyelia, five of tabes dorsalis, six of multiple sclerosis, four of peripheral neuritis, and one of a thalamic tumor. With the exception of one case of syringomyelia obtained from Dr. Tilney's private practice, they were patients from the Neurological Department of the Vanderbilt Clinic, and the diagnosis of each case was confirmed by the Chief of Clinic, Dr. McKendree. The experiments were made during 1917–1918, in the forenoons, in a room of the clinic, free from distraction, the temperature of which was kept between 22° and 25°C. As a rule, rather more preliminary practice was required with these patients than with normal subjects; enough was given to put the patient thoroughly at ease, and to give the experimenter an idea of the general type of response to be expected and a reasonable certainty that the patient understood the directions.

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[1] The experiment as originally planned covered four days, on each of which the double stimulus was given on forty areas of the body, and a single stimulus on twenty-six of these areas—the latter being equally divided between cold and hot, so that in four experiments, two measures for cold and hot respectively were obtained for each of the twenty-six areas, and four measures of double stimulation were obtained for forty areas. Owing to the difficulty of getting patients to return so often, the plan was varied so that three observations should give the same number of tests. On the first two days forty double stimulations were given as before, but the twenty-six areas were tested for *both* hot and cold. On the third day, two series of double stimulation were made, single stimuli being inserted only often enough to serve as a check. This was not a source of suggestion, however, because, as will be seen, all patients gave a considerable proportion of responses to double stimuli in which only one stimulus was perceived.

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Analysis of the cases will be made, on the basis of the tables and figures to be found at the end of the paper, the cases of each syndrome preceded by a summary of the findings for that syndrome. The various syndromes will then be compared, the general nature of the disturbance will be discussed, and the conclusions that seem justified will be drawn.

As a preliminary, however, it will be convenient to summarize the results of normal reactions in a form easily comparable with that in which abnormal cases will be presented.

I. GROSS DISTURBANCE.—While normal subjects show many individual differences in the general nature of their responses, nothing that may be called a gross disturbance appears. By this term is meant a striking disturbance shown by *failure to react;* or by *perverted response* which is a hot-cold response to a cold stimulus or a cold-hot response to a hot stimulus, as well as a hot response to a cold stimulus or a cold response to a hot stimulus.

II. GENERAL CHARACTER OF REACTION. *Type of Response to Double Stimulus.*—In spite of considerable fluctuation, normal subjects all show a majority of C–H responses, and a very small number of single responses.[2]

Range of Medians for Various Regions of the Body.—Among normals, no subject shows a range of more than .6 second for the median reaction-times of all regions, in any type of response. That is, a subject's reaction-time is fairly constant for all regions of the body.[3]

Extreme Reactions.—Since the measure used is the median and not the average time, it is not much affected by a few extremely long reactions. This is desirable, of course, in measuring the normal time, but in comparing abnormal cases with normal, it might cause the neglect of a number of extreme reactions, which might be of some importance. They can, therefore, be considered by themselves. Reactions of 3.0 seconds for separate stimuli or for the first interval of double responses do not occur among normals.[4] Reactions of 1.0 second or more as the second interval of double responses are rare, generally, though they occur in one subject in twenty-five per cent. of the cases.[5]

Consistency.—As it might be possible for a disturbance to be confined to a few areas, but at the same time to cause a large probable error in the measurement of the region including those areas, or a large fluctuation in the type of response for that region, the consistency of response was measured in the case of abnormal subjects (Infra, Table 8). For single stimuli, the measure used was the percentage of areas stimulated giving two correct responses—this is called consistency in type, and varies, for different normal subjects, from 85 to 100 per cent. for cold, and from 92 to 100 per cent. for hot. For double responses, the same measure was used—the percentage of areas stimulated giving two C–H responses, etc.; but this allowed more variation, as each area was stimulated four times. Consistency of C–H responses was high among normals, ranging for different subjects from 75 to 100 per cent.

The fluctuation of single areas in *time* might have been measured by the deviation of each area from its own average if there had been a sufficient number of cases, but it seemed best under the circumstances to take as a measure the range of each area having two similar responses. Thus, for normals, it was found that the percentage of areas which responded at least twice by C–H that had a very close range—less than .3 second—varied from 43 to 73 per cent. for the first interval, and from 45 to 80 per cent. for the second interval, for different individuals; while the percentage having a large range—1.0 second or more—was not more than 3 per cent. for the first interval, and varied from 0 to 29 per cent. for the second. In reaction-time, separate stimuli gave somewhat more consistent responses—68 to 96 per cent. of the areas having a small range for cold, 50 to

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[2] Neurological Bulletin I, p. 154.

[3] *Ibid.*, Tables 5–8 and 11, p. 145 *et seq.*

[4] *Ibid.*, Figs. 1, 2 and 4.

[5] *Ibid.*, Table 1, Subj. H, p. 141.

92 per cent. for hot, while the largest percentage having a range of 1.0 second or more was 7.6 per cent.

Diminution of Intensity of Sensation.—While subjects were not asked to differentiate degrees of intensity, there were a few instances among normals, and many among abnormals, when the sensation was reported as 'warm', instead of the usual 'hot'. Isolated instances of this sort might be due to sporadic fluctuations in the temperature of the blocks used for stimulus; but any considerable number of such cases would have to depend on subjective factors. Of course, if all responses should be 'warm', it might be explained as a language-habit of the patient; but if any distinction is made, it is fair to assume that it represents a subjective difference. Among normal subjects, no responses to a separate hot stimulus were of this sort; in the case of double stimulation, the highest percentage of warm responses was 7 per cent. There was no suggestion of any corresponding tendency to report 'cool' instead of 'cold'.

Intensification of Sensation.—It would be desirable to have a measure of this phenomenon also, but as any questioning by the experimenter would have been a distraction from the main purpose of the experiment and would in any case have been too open to suggestion to be very valuable, it is necessary to resort to collateral evidence. There is much less tendency to report increase in intensity, verbally, than decrease; partly, perhaps, because a very intense stimulus causes a reflex withdrawal which is itself an evidence of increase. This reflex is about the only measure we have of intensification, and it is of questionable value both because of large individual differences and because no record was kept of its appearance in normal subjects. It occurred, on abdomen or back in at least two subjects, but it was generally inhibited in the course of the preliminary practice.

SYRINGOMYELIA

Six cases were studied. In three, the disturbance was confined to one or a few regions; while in the other three there is a general disturbance, although in two of the latter, it is more marked on a few areas. The disturbance is shown by a marked lengthening of the reaction-time to single stimuli; by failure to perceive the stimulus at times; by a tendency to give perverted responses; by a higher percentage of single responses than normal to double stimuli and a long reaction-time in every type of response; by a large number of extreme reactions; by a more than normal inconsistency of response; and by a tendency to report the sensation as weak in intensity. Analyses of the cases follow, with references to the tables and figures at the end of the paper.

Case I.—P. K.; age 16; student; intelligence and coöperation good.

Onset.—Spinal curvature eleven years before test.

Sensation.—Not described in history.

I. Gross Disturbance.—Tables 6 and 7. No failure. Perversions occur on hand and arm to both cold and hot, but more often to cold.

II. General Character. *Type of Response.*—Fig. 1. Curve is practically normal for face and legs; but on arms, abdomen and back single responses to double stimuli are numerous:—H on arms; C on abdomen and back.

Median Reaction-time.—Table 1. In *single* stimuli C tends to be somewhat lengthened, except on face; while H is about normal except on arms and back. In *double* stimuli there is a general tendency toward lengthened reaction-time in the first interval of double responses; a marked similar tendency in the second interval on arms and back, and in the case of single responses.

Extreme Reactions.—Table 1, Fig. 6. *Single responses* show no more tendency to extreme reactions in double than in single stimuli, and are in both cases confined to cold. The tendency is more marked in double responses, and is more frequent for the second interval—slightly more frequent for H-C than for C-H type.

Consistency.—Table 8. In *single* stimuli there is a marked inconsistency in type of response for cold which is most evident on the right arm. The fluctuation in time, while greater than normal, is not very pronounced. In *double* stimuli no type of response occurs twice on half the areas, though C-H is the most frequent type. The fluctuation in time is greater than normal, and is particularly marked for the second interval of H-C responses. H ersponses fluctuate more than C.

Diminution of Sensation.—Table 9. Hot is diminished on all regions in a considerable percentage of cases—more in double than in single stimuli. Cold is somewhat diminished on arms.

Intensification.—Table 10. There is no reflex evidence, but patient reported that when sensation was 'sharp' distinction was difficult. He thought that it was cold that sent a shooting pain through region and that it 'got worse and worse' while warm 'stayed warm all the time'. Once he said: "I guess it's warm, because if it were cold I'd feel it." Perhaps this explains the number of times that C was reported as warm. It also implies an intensification of cold—though there is evidently a hypesthesia.

Single stimuli show cold to be more disturbed than hot: by its longer reaction-time; more extreme reactions; greater inconsistency in type and in time; and greater likelihood of perversion. But the results of double stimulation do not confirm this. The C-H type has at least a plurality of all responses; reaction to hot tends to be longer in double than in single stimuli, while to cold it remains about the same; and H responses are less consistent than C.

The disturbance is moderate in degree and is general rather than local, although face and legs escape.

Case II.—E. Wi. (colored); age 25; dental student; intelligence and coöperation good.

Onset.—About eighteen months before test.

Sensation.—Described in history, July, 1917: "Mild degree of disturbance to hot and cold." Frequently patient burned himself because "sensation was so delayed he did not feel pain in time."

I. Gross Disturbance.—Failure to react was consistent on left hand. Perversion occurred on right hand to hot, and on left foot to cold.

II. General Character. *Type of Response.*—Curve is altogether comparable with normal, except for the failures on left arm and the per cent. of single hot responses on neck.

Median Reaction-time.—In *single* stimuli the only lengthening is for hot, on left arm and back. In *double* stimuli, C-H responses are about normal, but H-C and H responses show some lengthening, particularly on left arm and abdomen.

Extreme Reactions.—The highest percentage of extreme reactions occurred for *single* responses to double stimuli, thus indicating a real disturbance for this type, both in the failure to perceive the double stimulus and in the slowing up of the response that did occur. In *double* responses the second interval is extreme more often than the first; but H is more frequently extreme than C, whether it is the first or second response.

Consistency. In *single* stimuli, H is less than normally consistent in type. The fluctuation in time is not excessive—though greater than normal—for either reaction. Both are very consistent (*i. e.*, they have a range of less than .3 sec.) in about half the areas. In *double* stimuli, no type occurs twice on fifty per cent. of the areas, though C-H approximates this. The fluctuation in time is greater than normal; and the deviation from the normal is greater in the C-H type than in the H-C.

Diminution of Sensation. Hot is diminished on all regions in a considerable number of cases—more in double than in single stimulation. The left arm, however, which showed the only gross disturbance, had relatively more reactions that were hot than had other regions. An initial perception of 'warm' followed by 'hot', from .3–2.0 seconds later, occurred a number of times but not with any regularity. 'Warm' was reported with temperatures even of 45° to 50°C. No weakening of the cold sensation was reported.

Intensification. No evidence, either reflex or verbal, was present.

Both single and double stimulations show more disturbance of the hot sensation than of the cold—more inconsistency in type; greater delay; more extreme reactions (at least in double responses); greater tendency to inhibition in double stimulation; and more tendency to diminished intensity.

Disturbance is marked locally, affecting, chiefly the left hand.

Case III.—A. C.; age 31; tailor; intelligence and coöperation fair.

Onset.—About nine years before test.

Sensation.—Described in history, June, 1917: "Normal to touch and apparently, also to pin-point. Thermal sense absent down to shoulder joints."

I. Gross Disturbance.—Failures were frequent or usual on arms and neck, and occurred on abdomen. Sensation was entirely lacking on back, except on shoulder blades. Perversion of cold was the rule on arms and occurred on abdomen; perversion of hot occurred on left arm and abdomen.

II. General Character. *Type of Response to Double Stimuli.*—Curve shows almost complete absence of double response; C type present only on face, neck, and abdomen; all other responses H.

Median Reaction-time.—In *single* stimuli, reactions are all very prolonged—hot more so than cold. In *double* stimuli the H type only occurs with any frequency, and is even slower than in separate stimuli.

Extreme Reactions.—About half the single responses to double stimuli and single stimuli are extreme. Double responses are very infrequent, but evidently tend to be relatively longer for the second interval than for the first.

Consistency.—As patient came only twice, while the experiment was planned for four days, there were not enough stimulations on each area to provide a measure for consistency. But consistency for single stimuli could not be very high for either stimulus, because of the number of failures and perversions, while for double stimuli, forty-two and five-tenths per cent. of the forty areas gave two hot responses, and fifty-three per cent. of these had a range of more than 1.0 second.

Diminution of Sensation.—Diminution of hot occurred to a considerable extent on arms and abdomen, and to some extent elsewhere. 'Warm' was reported with temperatures as high as 47°C. Diminution of cold occurred on face.

Intensification.—On legs, the double stimulus and the cold stimulus were several times accompanied by a marked reflex withdrawal. In one such case—to cold—patient said: "Cold or hot—something strong."

Both sensations are much disturbed. Cold is more often perverted and is more apt to cause a reflex movement; but although hot is more delayed and apparently more diminished, it takes most marked precedence in double stimulation.

The disturbance is marked generally over all regions, but is profound on arms and back.

Case IV.—L. A.; age 49; paper-hanger; intelligence and coöperation fair.

Onset.—About ten months before test.

Sensation.—Described in history, June, 1917: "Normal to pain and touch; cold felt as cold, but heat at times retarded and at times mistaken for cold on small finger and up ulnar side of forearm."

I. Gross Disturbance.—Failure to react occurred, but not persistently, on sole of each foot. Perversion of *cold* occurred on right forearm, right thigh, and right side of abdomen.

II. General Character. *Type of Response.*—Curve shows preponderance of double responses except on arms and face, with H-C type more frequent than normal. Single responses are frequent on arms and face and are present on legs; but do not occur on neck, abdomen or back.

Median Reaction-time.—Owing to the fact that patient came only twice, instead of four times, medians are not altogether reliable. For single stimuli, C seems to be a little slower than H—especially on face, left leg and abdomen, but times are not far from normal. For double stimuli, C-H responses are nearly normal, although face and legs seem to give rather long first interval and face a long second interval; H-C type tends to have more lengthened second intervals and generally longer first intervals; single responses are long.

Extreme Reactions.—The highest percentage of extreme reactions occurs for single responses to double stimuli, indicating, in connection with the long medians for this type, that these responses imply the most disturbance.

Consistency.—As in Case 3, consistency cannot be measured. It would be higher, with single stimuli, for H than for C, since the former had no errors. Results of double stimulation cannot be predicted; twelve and five-tenths per cent. of the areas gave two responses C-H, and an equal number gave two H-C.

Diminution of Sensation.—Hot stimulus was always reported 'warm'. Once, patient said: "Warm—good warm." As his English was somewhat limited, this may have been merely a language habit. No analogous *cool* was reported.

Intensification.—No evidence.

Contrary to the findings reported in the history, cold sensation seems to be more interfered with than hot, in both kinds of stimulation—it is slower separately and has more tendency to extreme reaction—in double responses as well as in single stimuli; only cold is perverted; and in double responses, hot takes precedence.

The disturbance is moderate and is general rather than local.

Case V.—J. Cl.; age 21; accountant; intelligence and coöperation good.

Onset.—About four years before test.

Sensation.—Described in history, July, 1915: "Touch, deep sensibility, and position sense O. K. Touch uncertain (*sic!*); heat and cold not differentiated over arms and upper chest."

I. Gross Disturbance.—Failure to react. Complete absence of sensation on upper extremities, for hot stimulus up to 52°; and for cold to 16°. Ice felt cold when grasped in hand, and cool on right forearm after several seconds. Perversion of cold was frequent, but in a number of cases a dubious correction was made. Indeed, patient was generally unable to tell, when he reported first one and then the other (whether the stimulus was double or single), whether both stimuli were present or whether he had first made a mistake—the second sensation seemed to take the place of the first.

II. General Character. *Type of Response.*—Curve shows marked predominance of H type, but greater frequency of C-H than of H-C. C responses are infrequent except on face.

Median Reaction-time.—Reaction-time is practically normal. In single stimuli C seems to be a trifle slower than H, and a little delayed on abdomen. In double stimuli, second interval is slightly long in double responses; single responses are prompt except in the case of C responses on back.

Extreme Reactions.—The only tendency to extreme reactions is in the case of the second interval of double responses, where half the reactions are over 1.0 second.

Consistency.—Except for the marked inconsistency in type of separate cold stimuli (due to the frequent perversions) and a larger than normal fluctuation for this type, patient's responses are very consistent in both type and time; but in double stimuli, it is the H type, not C-H, that is consistent.

Diminution of Sensation.—No diminution of sensation occurred in the first report; but twice on the cheeks, the double stimulus, although reported 'hot', evoked the comment: "It didn't feel either way afterward"—"it was just lukewarm afterward." A response of 'hot' was also occasionally followed by "a little cold at beginning."

Intensification.—There was no evidence of this.

Except for the anesthetic regions, hot seems to be intact; while cold shows some disturbance—its perversion; its slightly longer time; its inhibition in double stimuli; the fact that when the double stimulus was perceived, hot was more likely to follow when cold was felt first; and the few cases when cold was perceived without hot; are evidence of this.

The disturbance is complete locally, and is slight generally.

Case VI.—B. T. (female); age 22; stitcher; intelligence and coöperation fair.

Onset.—About eight years before test.

Sensation.—Described in history, October, 1917: "Over areas corresponding to sixth to eighth cervical segments of cord, on right side, there is diminished pain and temperature sensation, but touch is but slightly diminished."

I. Gross Disturbance.—Failure to react was the rule on left arm and was frequent on right arm. Perversion of cold was uniform on right arm.

II. General Character. *Type of Response.*—Curve is normal except on arms and face.

Median Reaction-time.—Number of cases is few, because patient came only once. Times seem to be somewhat longer than normal in all cases except the second interval of double responses. This may be due to insufficient adaptation to the task or is, perhaps, an individual characteristic. The longest times are for H responses to double stimuli.

Extreme Reactions.—Occur with frequency only in single responses to double stimuli.

Consistency.—Cannot be measured from one observation.

Diminution of Sensation.—All hot stimuli (temperatures 40–44) were called warm.

Intensification.—There was no evidence.

The perversion of cold and the fact that single responses to double stimuli were more often 'hot' than 'cold', suggest that cold is somewhat more disturbed than hot.

The disturbance is local and quite marked.

TABES DORSALIS

The five cases studied show some disturbance of a general character—marked in two; slight in two; and moderate in one. The disturbance is shown by some tendency toward perversion; by a marked deviation from the normal type of response; by a tendency toward somewhat lengthened reaction-times—especially with the double stimulus; by the number of extreme reactions; the inconsistency of response; and by a tendency toward intensification of the sensation. Analyses of the cases follow:

Case VII.—G. C.; age 39; barber; intelligence rather inferior.

Onset.—About two years before test.

Sensation.—Described in history, June, 1917: "Hypesthetic to pin-prick and to cotton wool."

I. Gross Disturbance.—No failure to react. Perversion of cold occurred consistently on lateral surface of both legs.

II. General Character. *Type of Response.*—Curve shows more single reactions than normal on every region except, perhaps, the face; being particularly high on legs, abdomen and back. H-C responses are slightly more frequent than C-H. There is a considerable number of doubtful responses.

Median Reaction-time.—In single stimuli all regions give lengthened times except neck, abdomen and back for H; and C is somewhat slower than H. In double stimuli, C-H responses show much lengthened first interval and long second interval on left leg and back. H-C responses are long in both intervals. Single responses are also long, H somewhat less than C.

Extreme Reactions.—Extreme reactions are frequent—more so in double than in single stimuli, and more for the first interval and for single responses than for the second interval. The tendency for both intervals is more evident in H-C than in C-H type.

Consistency.—Reaction to all kinds of stimulus is very inconsistent in type and fluctuating in time, except for the first interval of C-H responses.

Diminution of Sensation.—Diminution of hot occurred to some extent in both single and double stimuli; cold was diminished to a much greater degree than in other subjects, chiefly in double stimuli.

Intensification.—Some reflexes were present but were not very marked. They accompanied cold stimulation and double stimulation, and were mostly on abdomen and back.

Cold is evidently somewhat more disturbed than hot—it is more lengthened; shows some perversion; occurs somewhat less frequently than H as the first response; and is considerably diminished in intensity.

The disturbance is quite marked and is general rather than local.

Case VIII.—P. O.; age 36; railroad man; intelligence and coöperation good.

Onset.—About three years before test.

Sensation.—Described in history, May, 1917: "Touch and pain showed hypesthetic region on back, with bordering hyperesthetic area. Vibratory sensation diminished on lower extremities." (Temperature not mentioned.)

I. Gross Disturbance.—No failure to react. Perversion of hot on soles of feet; of cold on back.

II. General Character. *Type of Response.*—All are rather frequent. On the legs, C-H and C predominate; on neck and back, H.

Median Reaction-time.—In single stimuli medians are about normal, but H is somewhat quicker than C. In double stimuli the reaction is slower and more variable; first interval of C-H and second interval of H-C are about normal. H responses are generally slower than C.

Extreme Reactions.—In single stimuli extreme reactions are rare and occur only for C. Double stimuli increase the number of extreme reactions, particularly for single responses and for second interval of double. Hot, either as first or second response, is more apt to be extreme.

Consistency.—In single stimuli, consistency in type is normal for both, but fluctuation in time is marked for C. In double stimuli, no type gives even a moderate degree of consistency; in time, the fluctuation is greater than for single stimuli and is noticeably greater with C-H than with H-C responses.

Diminution of Sensation.—There is no evidence of this.

Intensification.—Reflex retractions, sometimes so pronounced as to interfere with stimulation, occurred on legs, abdomen and back, chiefly accompanying double stimulus. They were most violent on back, and increased rather than diminished as experiment progressed. Patient declared the stimulus felt 'scalding' and 'icy'. With separate stimuli often, and not infrequently with double, patient reported a second hotter or colder sensation from one to four seconds after first.

Although in single stimuli C is somewhat slower and more apt to be extreme than H—as well as more fluctuating—in double stimuli, the opposite is the case: hot is delayed more, whether as first or second response or whether it occurs alone, and is more apt to be extreme, although it is still less fluctuating than cold.

The disturbance is moderate and general, though less marked on upper extremities.

Case IX.—J. Co.; age 37; tailor; intelligence good, coöperation fair.

Onset.—About seven years before test.

Sensation.—Described in history, October, 1917: "Hypesthesia partial to pain and vibratory sensation."

I. Gross Disturbance.—Failure to react occurred, chiefly with hot stimulus on sole of left foot. Perversions of both stimuli occurred—of hot on face; more often of cold, on foot and back.

II. General Character. *Type of Response.*—Curve shows C-H responses in general to be most frequent; H-C very infrequent; C almost absent except on legs where they predominate; H very frequent on right arm, abdomen and back.

Median Reaction-time.—The only tendency toward lengthening of reaction-time occurs for the second interval of H-C responses.

Extreme Reactions.—The number of extreme reactions is small. It is greater for single stimuli than for double, except in the second interval, and occurs only with hot stimuli.

Consistency.—In single stimuli, consistency in both type and time is quite normal. In double responses, while no type has a very high consistency, the C-H type attains it in forty-five per cent. of the areas. The fluctuation in time is not great.

Diminution of Sensation.—There is a considerable tendency to report 'warm' instead of 'hot'—slightly greater in the case of double than of single stimuli. Twice, an initial perception of warm was followed by hot, not more than 1.5 seconds later. No diminution of cold was reported.

Intensification.—Stimulation on back was sometimes accompanied by a reflex, but the tendency was not marked, and there was no verbal comment.

The type of response suggests that on the legs the hot sensation is more interfered with than the cold; elsewhere there is not apparent a greater disturbance of one than the other, except in the rather general tendency toward a diminution of the intensity of hot.

The disturbance is very slight, but general.

Case X.—L. F. (female); age 53; cook; intelligence rather inferior; coöperation poor, influenced by suggestion.[6]

Onset.—About two years before test.

Sensation.—Described in history, September, 1917: "Normal."

I. Gross Disturbance.—No failure to react. Perversion of cold was general—the response usually H-C. This was evidently due, at least in part, to suggestibility and inference, as H-C responses were frequent to hot stimulus also. When error was pointed out to her on third day, the number of wrong responses was reduced to four, all of which occurred on arms and legs.

II. General Character. *Type of Response.*—Curve shows H-C type to be decidedly predominant, but its significance is questionable, as single stimuli also gave this response most frequently. Single C responses occur only on arms and legs; H only on abdomen and back.

Median Reaction-time.—In single stimuli, while there is much variability owing to the small number of correct responses, no significant tendency to long reaction-time is apparent. In double stimuli, the only lengthening occurs for the second interval of both C-H and H-C responses on the face, and in general for C responses.

Extreme Reactions.—There is no very marked tendency to extreme reaction. It is perhaps most significant in single responses to double stimuli; but while in single stimuli all extreme reactions are to hot, in single responses to double stimuli they are all to cold.

Consistency.—In spite of three trials instead of two for each area, single stimuli gave a low degree of consistency in type and a larger than normal fluctuation in time. Double stimuli—with three instead of four trials—were quite consistent in the H-C type. Fluctuation in time was not marked and was normal for second interval of both types.

Diminution of Sensation.—There is no evidence of this.

Intensification.—Reflexes are very numerous and occur on left arm and face as well as legs, abdomen and back. They occur more with single than with double stimulation and more with cold than hot. On abdomen, patient, asked which was more intense, called the cold (of 19°) 'worse' than the hot (44°).

One can hardly say that either sensation is more disturbed. At least a tendency for hot to take precedence, in spite of apparently greater intensification for cold, seems to be present.

There is evidently a slight general disturbance the real nature of which is, however, masked by patient's desire to 'do well'.

Case XI.—H. D.; age 49; metal lather; intelligence and coöperation good.

Onset.—About three years before test.

Sensation.—Described in history, June, 1917: O. K. (except blurring vision).

I. Gross Disturbance.—Neither failure nor perversion occurred.

II. General Character. *Type of Response.*—Curve shows a marked predominance of C, with infrequent double responses.

Median Reaction-time.—Reaction is generally quite slow. In single stimuli C is slower than H; but in double stimuli H-C and H are slower than C and C-H.

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[6] Patient was unduly impressed by instruction to react as quickly as possible, and evidently inferred that nearly all stimuli were double. She seemed to think the test a 'treatment' and to consider the stimulus more important than the response. On the third day her error in giving double responses to single stimuli was pointed out, and the error was reduced, though not eliminated, by her increased attention.

Extreme Reactions.—The tendency to extreme reactions is very marked in double stimuli, rather more when both stimuli are perceived. In all types the tendency is greater for hot than for cold, but this is not evident in single stimuli.

Consistency.—Single stimuli give perfectly consistent responses in type, and the fluctuation, though greater than normal, is not extreme. In double stimuli, C type is quite consistent; fluctuation is high, especially for double responses.

Diminution of Sensation.—There was some general diminution of hot, more in single than in double stimuli, and of cold on the left hand.

Intensification.—The abdomen and back gave, not infrequently, slight reflexes; but they were evidently to considerable extent inhibited. The sensation was extremely disagreeable, according to patient's statement, especially cold.

Although in single stimuli cold is somewhat slower than hot; in double stimuli the hot sensation is evidently more disturbed, as is shown by the greater frequency of the cold response, and the greater delay and greater tendency to extreme reactions in hot responses, whether in double responses or alone.

The disturbance is marked and general.

MULTIPLE SCLEROSIS

The six cases studied show slight disturbances which are general in four, and local in two. Disturbance is shown chiefly by deviation from the normal type of response to double stimuli; by lengthened second intervals of double responses; and by some tendency toward diminution of sensation. Analyses of the cases follow:

Case XII.—E. Wa. (female); age 43; housewife; intelligence good, coöperation excellent.

Onset.—About eighteen months before test (remission for months until a year previous).

Sensation.—Described in history, January, 1918: "Intact—pain, touch, temperature."

I. Gross Disturbance.—No failure to react. Perversion not present.

II. General Character. *Type of Response.*—The curve shows a tendency to predominance of H-C and H responses, particularly on upper part of body; the tendency is much less clear on legs, abdomen and back, and is rather strikingly reversed on the left leg.

Median Reaction-time.—In single stimuli medians are normal with slight variability, except, perhaps, on the neck and left leg for C, and abdomen for H, where there is some retardation. In double stimuli, double responses show a slight lengthening of first interval and a strikingly long second interval, the more remarkable, considering the promptness of reaction separately. C and H responses are delayed on legs and abdomen—H more than C.

Extreme Reactions.—The tendency toward extreme reactions in single stimuli, and in the first interval of double responses, is negligible; while for the second interval ninety per cent. of responses are 1.0 second or more. Single responses to double stimuli show the tendency to a small degree.

Consistency.—In single stimuli there is no inconsistency in type. The fluctuation in time, however, is rather more than normal. In double stimuli no type has a very great consistency—H being the most consistent. In time, double responses have not more than normal fluctuation in the first interval, but fluctuate markedly in the second.

Diminution of Sensation.—There is some diminution of hot, considerably more frequent in single than in double stimulation. In a considerable number of these cases, however, the initial perception of warm was followed by hot, from .8 to 4.6 seconds later. In several of these cases the interval was over 2.0 seconds, which is in itself quite unusual. No diminution of cold was reported.

Intensification.—There were no reflex retractions, although in this case, as in most multiple sclerosis cases, jerks of the legs were frequent—a random jerk, rather than an attempt to withdraw from the stimulus. Patient often reported, however, that sensation was a 'shock', which made the distinction difficult; but she asserted positively that it had no sting like very hot or very cold sensations.

Although hot tends to take precedence, except on legs and abdomen, it also shows a tendency to diminution and to an exaggeration of what may be called the crescendo effect (perception of warm followed by hot). It can hardly be said that one sensation is more disturbed than the other. What is chiefly disturbed is the immediate perception of the doubleness, regardless of which is perceived first.

The disturbance is slight and general, although it is more marked on legs and abdomen.

Case XIII.—J. Q.; age 25; occupation not stated; intelligence and coöperation fair.

Onset.—About three years before test.

Sensation.—Described in history, June, 1916: O. K. Dorsal roots were cut, July,

1916. Sensory examination, October, 1916, showed analgesia; temperature anesthesia on ventral surface of leg—left more than right; and hyperesthesia to cold on dorsal aspect of thighs and lateral part of legs.

I. Gross Disturbance.—Failure to react occurred once but was insignificant. Perversion was not present.

II. General Character. *Type of Response.*—The curve is quite fluctuating for different regions. It is to be noted that H-C responses do not occur; that the abdomen has a normal percentage of C-H responses; and that, except on face and neck, C is more frequent than H.

Median Reaction-time.—In single stimuli medians are prompt, with small variability—C slightly quicker than H. In double stimuli, C responses are normal whether alone, or as first interval; while hot is delayed both in H and in C-H responses.

Extreme Reactions.—The only tendency to extreme reactions was in second interval of double responses. Most marked on legs and back; least on abdomen.

Consistency.—Single stimuli give very consistent responses both in type and time. In double stimuli, C-H and C responses each occur twice on nearly half the areas. In time, the only fluctuation of extent is in the second interval of C-H responses.

Diminution of Sensation.—Hot stimulus was regularly reported 'warm'—even with temperatures up to 47°. As patient was English-speaking, and as response 'hot' did occur, this is probably not a mere language habit. No diminution of cold was reported.

Intensification.—Reflexes occurred with cold stimuli and with double stimuli to which response was 'cold' on back. (Temperature of 47° evoked no reflex.) Jerks of legs, but not withdrawals, also occurred.

While both sensations appear to be normal in single stimulation, double stimuli show some interference with the hot in the long second interval, and in the small number of cases in which hot occurs alone, as well as in the delay of such responses.

The disturbance is slight but general.

Case XIV.—G. P.; age 38; tailor; intelligence and coöperation fair.

Onset.—Four years before test.

Sensation.—Described in history, April, 1918: Normal.

I. Gross Disturbance.—No failure to react. One instance of perversion of hot occurred.

II. General Character. *Type of Response.*—Curve is practically normal—except on legs, where percentage of double responses falls and that of C rises. C is much more frequent than H.

Median Reaction-time.—In single stimuli medians are normal, with a normal amount of overlapping of C and H. In double stimuli, H-C type shows some lengthening of first interval, and of second interval on legs. Lengthening of second interval on legs is also present in C-H responses, which are otherwise normal. Single responses show some delay.

Extreme Reactions.—Extreme reactions are not very frequent and occur chiefly in second interval of double responses—more in H-C than in C-H.

Consistency.—In single stimuli consistency is normal, for both type and time. In double responses, the only abnormality in type is in the number of areas giving consistent C responses. In time the fluctuation is generally greater than normal, and is marked in the second interval of H-C responses.

Diminution of Sensation.—'Warm' was more frequent than 'hot'; and somewhat more frequent with double than with single stimuli. Occasionally, that is in three instances, the perception of warm was followed by hot—1.1, 1.5, and 2.0 seconds later. The only instance of diminution of cold occurred on the hand, where the response 'cold' followed 2.0 seconds after 'cool'.

Intensification.—There were frequent jerks of legs, but not a real withdrawal. On abdomen, however, patient sucked in his breath at the cold stimulus, and expressed surprise at its intensity.

The interference, in this case, is chiefly in the immediate perception of doubleness—whichever response came first—although when either was inhibited, it was more likely to be hot.

The disturbance is very slight, affecting chiefly the legs.

Case XV.—J. T.; age 19; student; intelligence and coöperation good.

Onset.—About eleven months before test.

Sensation.—Described in history, November, 1917: O. K.

I. Gross Disturbance.—No failure to react. Perversion not present.

II. General Character. *Type of Response.*—Curve shows H type to be most frequent, except on face, where C-H prevails; C type is very infrequent except on face.

Median Reaction-time.—In single stimuli times are normal; but are longer on legs than elsewhere. H seems to be a little quicker than C. In double stimuli, C-H responses show a delay only in the second interval on right leg; H-C responses are prolonged in both intervals on face, legs, and abdomen —and in second interval on right arm. Single responses are slow on legs.

Extreme Reactions.—There is some tendency toward extreme reactions, which is considerable only in the case of the second interval of double responses, especially of the H-C type.

Consistency.—In single stimuli there is no inconsistency in type, but rather more than normal fluctuation in time—especially for C. In double stimuli H type has a high degree of consistency, and C-H responses are fairly consistent. The fluctuation in time is not considerable except for H-C type, although C type shows much greater fluctuation than H.

Diminution of Sensation.—There is a slight degree of diminution of both sensations, more in the case of double than of single stimuli—chiefly on legs and face for hot, and on legs and arms for cold.

Intensification.—The only suggestion of this was two reflex withdrawals on back; and one verbal report of 'very hot'.

Single stimuli show scarcely any abnormality, but suggest a slight retardation of cold and a slightly greater variability. Double stimuli confirm the view that cold is more interfered with—it is likely to be absent; if present, it is much more likely to be followed by hot than to follow it; and it shows more fluctuation and more extreme reactions than hot either as the second interval or as a single response. It is interesting to note, in this connection, the number of uncertain responses, as when patient started to enunciate 'cold' but changed his mind; or said 'cold' and then thought it was a mistake. As in case of J. Cl., hot, when perceived, very often seems to displace cold.

The disturbance is moderate and general.

Case XVI.—I. R. (female); age 29; knitter; intelligence and coöperation good.

Onset.—About six years before test.

Sensation.—Dorsal roots were cut January, 1915—L II, III, V, S I. Examination in June, 1915, showed loss of sensations of touch, heat and cold over area L I and L II segments.

I. Gross Disturbance.—No failure to react. Perversions not present.

II. General Character. *Type of Response.*—Curve shows much apparent fluctuation between C-H and H-C type (number of cases was small as patient came only once) but is evidently normal, except for a larger proportion of single responses than normal on legs and back.

Median Reaction-time.—Single stimuli give perfectly normal times except on left leg, where both C and H show some delay. Double stimuli appear slightly to increase the time of the first interval, and very slightly the second interval—and more for C-H than for H-C responses, except on right arm.

Extreme Reactions.—Practically the only extreme reactions occurred in the second interval of double responses, more often in H-C than in C-H responses.

Consistency.—No measure, as there was only one observation.

Diminution of Sensation.—While there were no reports of 'warm', and only occasionally one of 'cool', patient said that sensation on legs 'felt funny . . . only a little . . . different from rest of body'.

Intensification.—Reflexes were frequent on legs and abdomen, but the sensation was not described as intense.

What slight disturbance there is seems to be about equal for both sensations.

There is a very slight disturbance, affecting chiefly the legs.

Case XVII.—J. B.; age 42; peddler; intelligence and coöperation good, but patient had only slight knowledge of English.

Onset.—About seven months before test.

Sensation.—Not noted in history, April, 1918.

I. Gross Disturbance.—No failure to react nor perversion.

II. General Character. *Type of Response.*—Reaction is quite uniformly C-H, except for single C responses on legs and back.

Median Reaction-time.—In single stimuli medians are normal; H is consistently slightly slower than C. In double stimuli the only tendency to delay is in the second interval, which is greater than 1.0 second for all regions except abdomen and back. The promptness of reaction otherwise makes this the more striking.

Extreme Reactions.—In single stimuli there were no extreme reactions; in double stimuli there were none except in the second interval where the majority were over 1.0 second.

Consistency.—No measure as patient came only once.

Diminution of Sensation.—All responses to hot stimulation were 'warm'; but whether this indicated a real diminution of the sensation or was due to patient's slight knowledge of English, could not be determined. No diminution of cold was reported.

Intensification.—Abdomen and back occasionally gave slight reflexes with cold stimulus.

While single stimuli gave normal reaction to both cold and hot, in double stimuli hot seemed to be somewhat interfered with—being markedly delayed in double responses; never occurring alone; and showing a tendency to be inhibited altogether on legs. (In preliminary practice, it tended to be inhibited also on left arm.)

The disturbance is very slight but general.

PERIPHERAL NEURITIS

In one of the four cases studied no disturbance is shown; in one, it is shown by the number of H–C responses, and single responses, and their increased reaction-time; and in two it could not be determined whether there was any disturbance or not. Analyses of the cases follow:

Case XVIII.—L. R.; age 17; student; intelligence and coöperation very good.

Onset.—November, 1916, median and ulnar nerves of right arm cut by glass.

Sensation.—Examination, January 10, 1917: Loss of touch and temperature (or deep hypesthesia) over palm and fingers, volar surfaces; and second and third phalanges of first, second and third fingers, dorsal surface. March 24, 1917: Whole palm and volar surface of fingers hypesthetic to cold —more on palm and first phalanges than on terminal phalanges. Palm normal to warm and hot. Hypesthesia to cold on dorsal surface of fingers.

Preliminary Tests.—April 27, 1918: Cold was more intense on median than on ulnar side of *right* palm—both sides being more sensitive than left palm. Second and third phalanges of first and second fingers of *right* hand were more sensitive to cold than left. Warm felt the same on both hands, but seemed a little slower in reaction on volar surface of fingers of right hand.

I. Gross Disturbance.—No failure; perversion not present.

II. General Character. *Type of Response.*—Curve is entirely normal for regions tested.

Median Reaction-time.—No difference is apparent in time of response for either arm; times are normal on those areas upon which sufficient tests were made to be reliable.

Extreme Reactions.—Rare.

Consistency.—In single stimuli consistency was normal in both type and time. In double stimuli, while consistency is normal in type, there is a slightly greater fluctuation in time. This is somewhat greater on right arm than on left—eighty-six per cent. of areas of left arm had a range under .3 second, fifty-six per cent. on right.

Diminution.—All responses to hot stimuli were 'warm'. (On right arm, temperature was 40° to 41°; on left, one series was 40° to 41°, another 45°. In one instance, sensation 'hot' followed .4 second later.) No diminution of cold was reported.

Intensification.—No evidence.

The method of the experiment shows no evidence of disturbance, although the introspective report of the patient in the preliminary tests shows that recovery is not yet quite complete.

Case XIX.—M. F.; age 19; student; intelligence and coöperation good.

Onset.—Median nerve of left arm cut by bullet wound, April, 1918.

Sensation.—Described in history, May, 1918: Hypalgesia and hypesthesia over volar surface of thumb and first and second fingers. Anesthesia over dorsal surface of first and second fingers.

Preliminary Tests.—May 18, 1918: Temperatures of 40° and 18° not felt on median surface of left hand and fingers, either dorsal or volar. Ice felt on dorsal, but not on volar median surface. Temperature of 45° felt slightly at times, on median palm and back of hand, but not on median volar surface of fingers.

I. Gross Disturbance.—Failures to react were consistent—to temperatures of 40° and 18° on dorsal and volar median surface of left hand and fingers, and to temperatures of 16° and 47° on volar median surface of fingers. No perversion occurred.

II. General Character. *Type of Response.*—Right arm shows normal distribution; left, a majority of H-C responses, and some single.

Median Reaction-time.—Tests are too few to be very reliable, but times are about normal, except for H-C responses on left arm, which are long in both intervals; single responses on left arm are also long.

Extreme Reactions.—Second interval of H-C responses gives a majority of reactions of 1.0 second or more. Single responses to double stimuli also give some extreme reactions.

Consistency.—Cannot be measured, as patient came only once; but where two measures of a kind were obtained for any area, the range was in no case as much as 1.0 second.

Diminution of Sensation.—Some diminution of both cold and hot occurred in double stimuli—more of cold than of hot, and more on left arm than on right.

Intensification.—No evidence.

The disturbance affects the entire left arm which shows more single responses, more H-C responses, more long reactions, and more diminution of intensity than the right. The deep hypesthesia, however, is restricted, and seems to be greater for cold than for hot.

CASE XX.—H. T. (female), age 20; bookkeeper; intelligence and coöperation very good.

ONSET.—Sudden paralysis of right side of face following abscess in right ear, February, 1917.

SENSATION.—Described in history, January, 1918: Normal except for loss of taste.

I. GROSS DISTURBANCE.—No failure to react. Perversion not present.

II. GENERAL CHARACTER. *Type of Response.*—The predominance of H-C responses on upper extremities and face of this patient led to a test of the legs also, where C-H were found to predominate. There were no single responses.

Median Reaction-time.—In single stimuli, medians are normally prompt, but H is consistently shorter than C. In double responses, the second interval is greater than normal, particularly in H-C responses.

Extreme Reactions.—These occurred only in second interval of double responses—in a considerable number of cases; more in H-C than in C-H type.

Consistency.—No measure, as there was only one observation, but the indications are that it would be high for the H-C type, since 47.8 per cent. of all areas which were stimulated twice with double stimuli gave two H-C responses.

Diminution of Sensation.—The only diminution occurred with hot stimuli on cheeks.

Intensification.—No evidence.

The predominance of H-C responses on upper extremities, and the long second interval, would certainly point to a disturbance of cold, if this were suspected. In the absence of any reason to suspect a disturbance, one must assume that it is an individual difference not shown among normal subjects tested. The absence of single responses also suggests normality.

CASE XXI.—B. P. (female); age 29; housewife; intelligence rather low; coöperation good.

ONSET.—Sudden paralysis of left side of face following pain in left ear consequent on exposure to cold, February 6, 1918.

SENSATION.—Described in history, February 11, 1918: "O. K. trifacial distribution."

I. GROSS DISTURBANCE.—No failure to react. Perversion not present.

II. GENERAL CHARACTER. *Type of Response.*—Right arm and face have more single responses than normal: H on arm and C on face. Left arm has normal distribution.

Median Reaction-time.—Medians are all longer than normal, except for second interval of C-H responses.

Extreme Reactions.—These occurred to a considerable extent in first interval of C-H responses and in single H responses; none on face.

Consistency.—No measure, as patient came only once.

Diminution of Sensation.—Diminution of hot occurred in a majority of responses on all regions—slightly more with double than with single stimulus. Once the perception of hot followed after 2.2 seconds.

Intensification.—No evidence.

In the absence of more than one observation, particularly with a patient of limited intelligence, it would be rash to assert whether or not the long reaction-times, and the single responses on the right arm as well as on the face, indicated a disturbance on either region. Unfortunately, a second observation could not be obtained.

CAPSULO-THALAMIC LESION

CASE XXII.—D. L.; deaf mute; age 16; intelligence and coöperation very good in spite of his incapacity. (Patient was able to articulate sufficiently to make verbal responses though, of course, more slowly than normal.)

ONSET.—About two years before test.

SENSATION.—Described in history, October 1917: "Hypersensitivity to pain and light touch on right side of body." No mention of temperature.

I. Gross Disturbance.—There were some failures with hot stimuli on right arm and on right side of back; and with double stimuli on right arm. Perversion of hot is almost complete on right side of body except on face, where perversion of cold occurred. In order to see whether patient was possibly getting paradoxical cold sensations, he was tested with temperatures of 36° to 37°. This also was called 'cold' on the right side, and 'rather hot' on left.

II. General Character. *Type of Response.*—C predominates on right side (hot not felt even alone) but C-H responses are normally frequent (or nearly so) on the left side. Single responses are rather more frequent than usual on all regions.

Median Reaction-time.—Reaction is generally longer than normal, but allowance must be made for the speech defect. They are fairly regular, however, and in H-C responses, the second interval is almost normal.

Extreme Reactions.—In spite of the generally long reaction-time there are few extreme reactions in single stimuli. In double stimuli the tendency is greater, particularly for cold in the first interval or alone, and in the second interval; oftener in C-H than in H-C.

Consistency.—In single stimuli, there is considerable inconsistency in type with hot stimuli; and fluctuation in time with both. In double stimuli, no type reaches normal consistency; but C-H and C responses occur twice with a fair degree of frequency. Fluctuation in time is fairly high.

Diminution of Sensation.—No evidence.

Intensification.—Reflexes were frequent on right side of body—even on the arm—for both sorts of stimuli and sensation was rather painful.

The disturbance of hot is profound on the entire right side of body; but allowing for the speech defect, the left side is probably about normal.

[EXPLANATION OF TABLES WILL BE FOUND ON PAGE 372]

TABLE 1

In this and the following tables, the regions are numbered from I–VIII as follows: right arm, left arm, face, dorsal surface of neck, right leg, left leg, abdomen, back.

MEDIAN REACTION-TIME (IN SECONDS) FOR SEPARATE AND DOUBLE STIMULI IN SYRINGOMYELIA

Case I—P. K.

	Separate Cold Stimuli				Double Stimuli										
					Response C–H							Response Cold			
			No. of Cases		1st Interval		2d Interval		No. of Cases					No. of Cases	
	Med.	P. E.	3.0′+	Total	Med.	P. E.	Med.	P. E.	1st Int. 3.0′+	2d Int. 1.0′+	Total	Med.	P. E.	3.0′+	Total
I	2.4	1.7–3.1*	1	2	1.4	.25	2.0	.75	2	6	9	2.1	1.4–2.8		2
II	1.8	.25	1	11	2.4	.45	3.5	1.50	2	3	5	2.5	.70	2	10
III	1.2	.00		5	1.8	.40	.5	.10		1	7	1.8	1.7–1.9		2
IV	1.5			2											
V	1.7	.30		5	1.8	.20	.5	.25		3	15				
VI	1.8	.05		5	1.4	.30	.6	.30		4	10	3.0	2.2–3.8	1	2
VII	1.9	1.6–5.4	1	3	1.5	.25	.9	1.20	1	2	5	2.1	.35		4
VIII	1.5	.20		5	2.2	1.8–2.6	1.6	1.20		1	2	1.8	.20		7
Totals			3	38					5	20	53			3	27
	Separate Hot				Response H–C							Response Hot			
I	1.6	.35		11	1.5	1.2–1.9	2.5	.8–4.2		1	2	2.1	.35		18
II	2.0	.30		13	2.8	.50	1.9	1.50	2	4	6	2.2	.50		10
III	1.4	.20		7	2.2	1.4–3.4	.5	.4–3.6	1	1	3				
IV	1.5	1.2–1.8		2								1.1			1
V	1.3	.05		6								1.9	1.8–2.0		3
VI	1.5	.20		6	2.2	.05	.6	.20		1	4	1.9	1.2–2.7		2
VII	1.4	1.3–2.7		4	1.5	1.2–1.8	.4	.3–.5			2	2.5			1
VIII	1.8	.15		4	1.6		6.0			1	1	2.0	1.6–2.4		2
Totals				53					3	8	18				37

* Total range is given with less than four cases.

TABLE I. (Syringomyelia continued)

Case II—E. Wi.

	Separate Cold Stimuli				Double Stimuli										
					Response C–H							Response Cold			
	Med.	P. E.	No. of Cases		1st Interval		2d Interval		No. of Cases			Med.	P. E.	No. of Cases	
			3.0′+	Total	Med.	P. E.	Med.	P. E.	1st Int. 3.0′+	2d Int. 1.0′+	Total			3.0′+	Total
I	1.2	.25		12	1.3	.20	.4	.05	1	1	24				
II	1.3	.95	2	12	1.1	.20	.5	.10		1	7	3.3	1.75	4	6
III	1.2	.20		6	1.2	.20	.4	.20		2	8	1.0			1
IV	1.1	.9–1.3		2											
V	1.1	.20		6	1.6	.25	.7	.80	1	6	16	1.4	1.3–1.7		3
VI	1.2	.00		5	1.3	.25	.8	.50		7	16	1.6	.25	1	8
VII	.9	.05		4	1.3	.25	.6	.20		2	12				
VIII	1.1	.10		4	1.1	.15	.4	.05			9	.9			1
Totals			2	51					2	19	92			5	19
	Separate Hot				Response H–C							Response Hot			
I	1.1	.20	1	9	1.7	.25	.4	.05			7	2.1			1
II	2.0	1.00	2	7	2.5	1.6–3.4	.8	.6–1.1	1	1	2	3.7		1	1
III	1.0	.10		6	1.2	.10	1.1	.10		3	4	1.2			1
IV	1.0	.9–1.2		2								1.4	1.4–1.4		2
V	1.3	.10		6	1.7	.10	.6	.05			4	1.3			1
VI	1.3	.15		5											
VII	1.0	.05		4	1.6	1.1–2.1	1.2	.5–2.0		1	2	2.8	1.2–4.4	1	2
VIII	2.2	1.1–2.7		3	1.3	.25	.4	.05		1	6				
Totals			3	42					1	6	25			2	8

Case III—A. C.

	Separate Cold Stimuli				Double Stimuli										
					Response C–H							Response Cold			
	Med.	P. E.	No. of Cases		1st Interval		2d Interval		No. of Cases			Med.	P. E.	No. of Cases	
			3.0′+	Total	Med.	P. E.	Med.	P. E.	1st Int. 3.0′+	2d Int. 1.0′+	Total			3.0′+	Total
I	3.0		1	1											
II	3.1	2.4–3.8*	1	2											
III	1.5	1.3–1.6		3								2.2			1
IV												4.6		1	1
V	3.1	2.0–4.8	2	3	2.6	2.5–2.7	3.2	1.5–5.0		2	2				
VI	1.7	1.3–4.3	1	3											
VII	3.9	3.8–4.1	2	2								2.9	1.6–4.3	1	3
VIII	†3.2	2.7–3.8	1	2											
Totals			8	16						2	2			2	5
	Separate Hot				Response H–C							Response Hot			
I	4.5	.90	4	4								4.8	1.35	5	6
II	4.9	2.8–5.2	2	3								7.5	6.4–8.6	2	2
III	2.6	1.4–3.0	1	3	2.2		3.6			1	1	2.0	.00		4
IV												9.6		1	1
V	2.1	1.6–2.4		3								2.6	.60	4	10
VI	2.0	1.6–2.8		3								2.2	.30	2	12
VII	4.2	2.9–5.5	1	2								4.0	.30	5	5
VIII	†2.1	1.8–2.4		2								4.1	.90	3	4
Totals			8	20						1	1			22	44

* Total range given with less than four cases.

† Areas stimulated on back were shoulder blades, since usual areas did not respond at all.

TABLE I. (Syringomyelia continued)

Case IV—L. A.

	Separate Cold Stimuli				Double Stimuli										
					Response C–H							Response Cold			
			No. of Cases		1st Interval		2d Interval		No. of Cases					No. of Cases	
	Med.	P. E.	3.0"+	Total	Med.	P. E.	Med.	P. E.	1st Int. 3.0"+	2d Int. 1.0"+	Total	Med.	P. E.	3.0"+	Total
I	1.5	.10		5	1.0	.8–1.2	.6	.5–.7			2	3.0	1.3–4.0	2	3
II	1.7	.15	1	6	1.4	1.4–2.4	.3	.3–.5			3	1.7	1.7–2.0		3
III	2.2	1.3–5.1	1	3	1.8	1.6–2.0	2.1	.6–3.6		1	2	2.2	2.0–2.5		2
IV	1.7			1											
V	1.3	1.3–1.4		2	1.8	.25	.4	.05		1	7	2.9			1
VI	2.2	1.5–2.4		3	1.9	.30	.5	.05		1	4	1.8			1
VII	2.5	2.4–2.6		2	1.5	.10	.4	.10			5				
VIII	1.3			1	1.5	1.4–1.7	.4	.4–.4			2				
Totals			2	23						3	25			2	10
	Separate Hot				Response H–C							Response Hot			
I	1.4	.40		6	1.8	.60	.8	.15		2	6	2.1	.75	1	5
II	1.5	.05		6	1.8	.30	.5	.15	1	1	7	1.7	1.0–2.0		3
III	1.2	1.1–1.5		3								2.0	1.1–2.9		2
IV	1.8			1	1.6	1.5–1.7	2.6	2.6–2.7		2	2				
V	1.7	1.6–2.9		3	2.0	1.9–2.2	.6	.5–.7			2	4.0		1	1
VI	1.6	.9–1.9		3	1.8	.10	1.3	.50		2	5	2.6	2.2–3.1	1	2
VII	2.0	1.6–2.5		2	1.6	1.2–2.4	.4	.4–2.8		1	3				
VIII	2.1	1.4–2.8		2	1.6	.20	.4	.05			6				
Totals				26					1	8	31			3	13-

Case V—J Cl.

	Separate Cold Stimuli				Double Stimuli										
					Response C–H							Response Cold			
			No. of Cases		1st Interval		2d Interval		No. of Cases					No. of Cases	
	Med.	P. E.	3.0"+	Total	Med.	P. E.	Med.	P. E.	1st Int. 3.0"+	2d Int. 1.0"+	Total	Med.	P. E.	3.0"+	Total
I															
II															
III	1.0	.15		7	.8		1.6			1	1	1.1	1.0–1.3		2
IV	1.2	1.0–1.2*		3								1.3	1.1–1.6		2
V	1.2	.05		12	1.2	.05	.9	.15		3	6				
VI	1.2	.05		4	1.2	.05	.7	.45		3	8				
VII	1.7	1.6–1.8		2								1.1	.9–1.3		2
VIII	.9	.8–1.1		2								2.2	2.1–2.3		2
Totals				30						7	15				8
	Separate Hot				Response H–C							Response Hot			
I															
II															
III	.9	.05		9	1.6		1.7			1	1	1.0	.05		8
IV	.9	.9–.9		2								1.1	1.1–1.2		2
V	1.1	.10		7								1.4	.10		18
VI	1.1	.10		7								1.4	.10		16
VII	1.0	.15		8								1.2	.20		14
VIII	1.0	.10		9								1.2	.25		14
Totals				42						1	1				72

* Total range given with less than four cases.

TABLE I. (Syringomyelia concluded)

Case VI—B. T.†

	Separate Cold Stimuli				Double Stimuli										
					Response C–H							Response Cold			
	Med.	P. E.	No. of Cases		1st Interval		2d Interval		No. of Cases			Med.	P. E.	No. of Cases	
			3.0″+	Total	Med.	P. E.	Med.	P. E.	1st Int. 3.0″+	2d Int. 1.0″	Total			3.0″+	Total
I												2.2	1.6–2.9		3
II	4.4		1	1											
III	2.1			1	1.7		.4				1				
IV	2.0			1											
V	1.7	1.5–2.0		2	1.9	.25	.4	.05		1	5				
VI	2.4			1	1.8	.25	.5	.00			4	2.4			1
VII	1.3			1	1.4	1.2–1.8	.4	.4–.4			3				
VIII	2.1			1	1.7	1.7–1.9	.4	.4–.5			3				
Totals			1	8						1	16				4
	Separate Hot				Response H–C							Response Hot			
I	1.7	1.7–2.8		3								3.3	2.1–4.5	1	2
II	1.8			1								2.3	1.9–4.8	1	3
III	1.9	1.8–2.1		2	2.2		.5				1	1.6			1
IV	(No	stimuli)													
V	2.6			1								3.0		1	1
VI	2.4	2.2–2.6		2	3.8		.7		1		1				
VII	1.8			1	2.4		.8				1				
VIII	(No	stimuli)										2.4			1
Totals				10					1		3			3	8

TABLE 2

MEDIAN REACTION-TIME FOR SEPARATE AND DOUBLE STIMULI IN TABES DORSALIS

Case VII—G. C.

	Separate Cold Stimuli				Double Stimuli										
					Response C–H							Response Cold			
	Med.	P. E.	No. of Cases		1st Interval		2d Interval		No. of Cases			Med.	P. E.	No. of Cases	
			3.0″+	Total	Med.	P. E.	Med.	P. E.	1st Int. 3.0″+	2d Int. 1.0″+	Total			3.0″+	Total
I	2.3	.35	2	13	3.4	.50	.7	.40	6	2	7	2.6	.65	3	7
II	2.3	.40	1	13	2.6	.30	.5	.15	2		13	3.0	.20	2	4
III	2.1	.20	1	6	} 2.8	.35	.6	.45	3	1	7	4.4		1	1
IV	1.8	1.6–2.0*		2											
V	2.1	.30		4	3.4	2.8–3.5	.6	.6–.8	2		3	3.0	3.0–3.1	2	2
VI	2.6	.35	1	4	3.1	2.6–4.6	1.7	1.2–2.0	2	3	3	3.9	.80	3	4
VII	2.0	2.0–2.2		3											
VIII	1.6	.15		4	3.9	.25	1.0	.20	3	2	4	3.6	.25	5	5
Totals			5	49					18	8	37			16	23
	Separate Hot				Response H–C							Response Hot			
I	2.0	.50	1	13	2.8	.25	.6	.15	5	2	12	2.0	.50	1	5
II	2.0	.20		13	3.1	.65	1.0	.30	6	5	18	2.9	1.4–3.7	1	3
III	1.9	.10		4	} 2.8	.55	.7	.15	2	2	5				
IV	1.3	1.1–1.6		2								1.6	1.5–1.8		2
V	2.5	.40	2	7	4.1	.75	2.0	1.40	4	2	4	3.6	.65	6	10
VI	3.1	.75	4	6	3.3	.25	2.1	.30	4	3	4	3.1	.30	5	9
VII	1.4	.00		4	2.4	.40	.9	.75	3	5	10	2.2	1.3–2.2		3
VIII	1.3	.05		4	2.9	.30	.7	.10	2	1	4	1.2	.9–2.0		3
Totals			7	53					26	20	57			13	35

† B. T. came only *once;* as the test was planned for *four* observations, the number is too small to make conclusions more than suggestive.

* Total range given with less than four cases.

TABLE 2. (Tabes Dorsalis continued)

Case VIII—P. O.

	Separate Cold Stimuli				Double Stimuli										
					Response C–H							Response Cold			
	Med.	P. E.	No. of Cases		1st Interval		2d Interval		No. of Cases			Med.	P. E.	No. of Cases	
			3.0″+	Total	Med.	P. E.	Med.	P. E.	1st Int. 3.0″+	2d Int. 1.0″+	Total			3.0″+	Total
I	1.2	.00	1	13	1.5	.50	.8	.50	1	5	11	3.0	.35	4	7
II	1.0	.10		12	1.3	.30	2.5	.55	1	6	8	1.0	.20	1	5
III	1.3	.30		6	1.4	.15	1.1	.55		3	5	1.1	.05		4
IV	2.1	.8–3.4	1	2											
V	1.0	.05		6	1.7	.40	1.0	.50	2	6	12	1.4	.20	2	9
VI	1.2	.25		6	1.6	.10	1.0	.50		3	8	1.6	.40	2	11
VII	1.1	.40		4	1.2	.40	.5	.00			4	6.6	1.6–8.6	2	3
VIII	.8	.5–.8		3	1.7	1.3–2.2	2.2	.6–3.8		1	2	1.7	.05	1	5
Totals			2	52					4	24	50			12	44
	Separate Hot				Response H–C							Response Hot			
I	1.2	.00		12	1.8	.35	.5	.55	1	3	9	1.5	.25		6
II	.9	.10		12	1.4	.45	.5	.65	1	5	12	2.4	.70	4	6
III	1.1	.15		6	1.6	.05	1.0	.60	2	2	4	2.4			1
IV	.6	.6–.7		2								1.3	1.2–1.4		2
V	1.0	.05		6	1.6	1.6–1.7	.3	.3–.4			2	5.6		1	1
VI	1.2	.10		6	2.0	2.0–2.3	.4	.4–.5	1		2	1.9	1.3–4.6	1	3
VII	1.0	.00		4	1.6	.45	.4	.05		1	6	1.7	1.3–3.2	1	3
VIII	.9	.05		4	2.0		.8				1	1.4	.20		9
Totals				52					5	11	36			7	31

Case IX—J. Co.

	Separate Cold Stimuli				Double Stimuli										
					Response C–H							Response Cold			
	Med.	P. E.	No. of Cases		1st Interval		2d Interval		No. of Cases			Med.	P. E.	No. of Cases	
			3.0″+	Total	Med.	P. E.	Med.	P. E.	1st Int. 3.0″+	2d Int. 1.0″+	Total			3.0″+	Total
I	1.0	.15		14	1.1	.05	.4	.00			5	1.5	.20		7
II	1.0	.10		14	1.3	.10	.5	.05		1	16	.9	.9–1.0		2
III	1.0	.15		7	1.4	.10	.6	.05		1	9				
IV	1.0	.9–1.2*		2											
V	1.1	.20		6	1.4	.15	.7	.40		3	10	1.3	.15		12
VI	1.1	.10		6	1.2	.20	.6	.20		2	6	1.3	.20		16
VII	1.0	.00		4	1.1	.15	.5	.15	1	1	6	6.8		1	1
VIII	1.0	1.0–1.6		3	1.4	.25	.5	.10		1	5	1.3	1.3–1.4		2
Totals				56					1	9	57			1	40
	Separate Hot				Response H–C							Response Hot			
I	1.1	.10		12								1.3	.10		20
II	1.1	.10		14	1.3	.05	.3	.10		1	8	1.3	.20		5
III	1.0	.05		6	1.2	1.1–1.4	2.4	2.2–2.7		2	2	1.0	.05		4
IV	1.0	1.0–1.0		2								1.4			1
V	1.7	.75	2	7	1.6		1.0			1	1	2.7			1
VI	1.3	.10	1	5								1.2			1
VII	1.4	.20	1	5	1.7		.9				1	1.4	.20	1	8
VIII	1.5	.15		4	1.5	1.4–1.6	1.0	.5–1.6		1	2	1.5	.25		8
Totals			4	55						5	14			1	48

* Total range given with less than four cases.

TABLE 2. (Tabes Dorsalis concluded)

Case X—L. F.

	Separate Cold Stimuli				Double Stimuli										
					Response C–H							Response Cold			
	Med.	P. E.	No. of Cases		1st Interval		2d Interval		No. of Cases			Med.	P. E.	No. of Cases	
			3.0′+	Total	Med.	P. E.	Med.	P. E.	1st Int. 3.0′+	2d Int. 1.0′+	Total			3.0′+	Total
I	1.0	.30		9	1.2	.30	.6	.15			7	1.6	1.00	2	6
II	1.6	.65		8	1.6	.40	.6	.10		1	5	1.7	.25		6
III	1.0	.25		6	2.6	2.4–2.8	1.4	.4–2.5		1	2				
IV	1.0	.8–1.2		2											
V	1.4	1.3–1.7		3	.9	.8–1.0	.4	.4–.5			2	2.1	.00		4
VI	1.5	1.0–1.5		3	1.0	.7–1.2	.3	.3–.4			3	1.9	1.4–2.2		3
VII	1.0	.8–1.1		3											
VIII	1.9	1.8–2.1		2	1.8		.8				1				
Totals				36						2	20			2	19
	Separate Hot				Response H–C							Response Hot			
I	1.4	.60	1	16	1.6	.15	.4	.05		2	11				
II	1.2	.50		10	1.4	.30	.4	.05		2	14				
III	.9	.05		6	1.1	.15	1.3	.75		6	10				
IV	1.1	.9–1.4		2											
V	2.3	1.6–3.0	1	2	1.1	.30	.4	.05		1	12				
VI	1.7	.00	1	4	1.0	.40	.4	.05			12				
VII	1.1	1.0–1.1		3	.9	.05	.4	.05		1	8	1.0	.15		4
VIII	1.1	1.1–1.2		3	.8	.25	.4	.10		1	7	.8	.05		4
Totals			3	46						13	74				8

Case XI—H. D.

	Separate Cold Stimuli				Double Stimuli										
					Response C–H							Response Cold			
	Med.	P. E.	No. of Cases		1st Interval		2d Interval		No. of Cases			Med.	P. E.	No. of Cases	
			3.0′+	Total	Med.	P. E.	Med.	P. E.	1st Int. 3.0′+	2d Int. 1.0′+	Total			3.0′+	Total
I	1.6	.35	1	13	1.4	.10	1.5	1.15		3	5	2.6	.50	3	9
II	1.9	.20	1	12	3.2		1.8		1	1	1	2.0	.85	4	13
III	1.6	.20		6	2.0		2.7			1	1	1.8	.20	1	7
IV	2.2	1.6–2.9*		2								3.2		1	1
V	1.8	.20	1	6								2.1	.25	2	12
VI	2.1	.10		6								2.6	.35	3	13
VII	1.7	.40		5	3.6	1.9–5.4	2.1	1.6–2.6	1	2	2	2.4	.55	3	9
VIII	2.3	.10		4								2.8	.70	4	10
Totals			3	54					2	7	9			21	74
	Separate Hot				Response H–C							Response Hot			
I	1.6	.30		12	7.3		.5		1		1	2.2	.45	2	9
II	1.7	.20		12	3.7	3.7–3.8	2.6	.5–4.8	2	1	2	3.5	1.15	4	8
III	2.3	.70		6	4.4		3.6		1	1	1				
IV	1.6	1.3–1.9		2								2.7	2.7–2.8		2
V	2.7	.90	2	5	3.2		4.3		1	1	1	3.0	.65	3	5
VI	2.0	.45	1	6								3.5	.70	3	5
VII	1.6	.25		5								4.9	3.7–6.6	2	2
VIII	1.3	.05		4								2.9	2.0–3.8	1	2
Totals			3	52					5	3	5			15	33

* Total range given with less than four cases.

TABLE 3

MEDIAN REACTION-TIME FOR SEPARATE AND DOUBLE STIMULI IN MULTIPLE SCLEROSIS

Case XII—E. Wa.

	Separate Cold Stimuli				Double Stimuli										
					Response H–C							Response Cold			
	Med.	P. E.	No. of Cases		1st Interval		2d Interval		No. of Cases			Med.	P. E.	No. of Cases	
			3.0″+	Total	Med.	P. E.	Med.	P. E.	1st Int. 3.0″+	2d Int. 1.0″+	Total			3.0″+	Total
I	1.2	.10		14	1.6	1.4–2.4	3.3	1.2–4.4		3	3				
II	1.2	.10		12	1.6	.15	1.2	2.20		3	5	1.2			1
III	1.3	.50		7											
IV	1.6	1.5–1.7*		2											
V	1.2	.10		6	1.4	.20	1.4	2.50		6	7	1.5	.20		4
VI	1.6	.20		7	1.5	.25	2.7	1.50		5	5	1.5	.25	1	12
VII	1.3	.05		4	1.9	1.4–2.5	2.4	2.2–4.8		3	3	1.7	1.5–3.2	1	3
VIII	1.2	.10		4	1.4	.05	1.4	.85		2	4	1.2			4
Totals				56						22	27			2	24
	Separate Hot				Response H–C							Response Hot			
I	1.3	.10		12	1.4	.25	3.0	1.25		10	10	1.4	.20		19
II	1.2	.10		12	1.3	.20	2.7	1.45		10	10	1.4	.20	1	17
III	1.2	.25		6	1.4	.25	2.4	1.25		8	8	1.5	.10		4
IV	1.2	1.1–1.3		2								1.3			1
V	1.4	.15		7	1.8	1.6–2.1	5.9	4.8–7.0		2	2	2.0	.15	1	12
VI	1.4	.25	1	6	1.6		6.1			1	1	2.6	1.00	3	6
VII	1.7	.10		4	1.7	1.6–5.6	1.3	1.0–1.9	1	3	3	2.2	1.8–2.4		3
VIII	1.4	.05		4	1.4	.20	2.8	1.65		5	7	1.3			1
Totals			1	53					1	39	41			5	60

Case XIII—J. Q.

	Separate Cold Stimuli				Double Stimuli										
					Response C–H							Response Cold			
	Med.	P. E.	No. of Cases		1st Interval		2d Interval		No. of Cases			Med.	P. E.	No. of Cases	
			3.0″+	Total	Med.	P. E.	Med.	P. E.	1st Int. 3.0″+	2d Int. 1.0″+	Total			3.0″+	Total
I	.9	.10		12	.9	.10	.8	.45		9	19	.9	.05		8
II	.8	.05		12	.9	.20	.8	.20		4	11	.9	.10		16
III	.9	.10		6	.8	.05	1.6	.75		3	5	.8	.8–1.1		3
IV	1.1	1.0–1.3		2											
V	1.3	.10		6	1.1	.00	1.5	.20		4	4	1.2	.10		14
VI	1.2	.00		6	1.1	.10	1.6	.80		7	10	1.2	.05		10
VII	.8	.05		4	.8	.05	.6	.30		4	14	.8	.8–.8		2
VIII	.9	.15		4	.8	.6–1.0	3.1	2.5–3.8		2	2	.8	.00		14
Totals				52						33	65				67
	Separate Hot				Response H–C							Response Hot			
I	.9	.05		12								1.5	.20		6
II	1.0	.05		12								1.3	.15		5
III	.8	.05		6								1.0	.10		5
IV	1.0	.9–1.1		2								1.4	1.0–1.5		3
V	1.4	.20		8								1.6	.55	1	5
VI	1.4	.40		7								2.4	2.4–2.6		3
VII	1.2	.05		4											
VIII	1.1	.05		4											
Totals				55										1	27

* Total range given with less than four cases.

TABLE 3. (Multiple Sclerosis continued)

Case XIV—G. P.

	Separate Cold Stimuli				Double Stimuli										
					Response C–H							Response Cold			
	Med.	P. E.	No. of Cases		1st Interval		2d Interval		No. of Cases			Med.	P. E.	No. of Cases	
			3.0″+	Total	Med.	P. E.	Med.	P. E.	1st Int. 3.0″+	2d Int. 1.0″+	Total			3.0″+	Total
I	1.3	.10		14	1.4	.05	.5	.30		6	22	1.9	1.3–2.8		3
II	1.2	.10		13	1.3	.35	.5	.10		3	20	1.7			1
III	1.2	.00		6	1.4	.15	.4	.10		3	15				
IV	1.5	1.2–1.8*		2											
V	1.6	.45		6	1.9	.50	.6	.40	2	3	9	1.7	.50		12
VI	1.3	.30		5	1.6	.15	1.0	.30		4	7	1.8	.20		15
VII	1.2	1.0–2.0		3	1.4	.30	.5	.15		2	4	1.3	1.1–1.6		2
VIII	1.3	.15		4	1.4	.15	.6	.15		4	10	1.6	1.2–1.6		3
Totals				53					2	25	87				36
	Separate Hot				Response H–C							Response Hot			
I	1.3	.15		13	1.8	1.7–2.0	.5	.5–.6			2	2.0	.10		5
II	1.2	.10		13	1.6	.30	.9	.60		5	11				
III	1.2	.20		7	2.1		.4				1				
IV	1.3	1.2–1.4		2											
V	1.4	.10		5	2.1	2.0–3.0	1.6	1.1–3.4	1	3	3				
VI	1.4	.35		7	1.8	1.7–2.0	2.7	.7–4.7		1	2				
VII	1.3	.05		5								1.8			1
VIII	1.5	.00		4	1.7	1.4–2.1	.5	.5–1.2		1	3				
Totals				56					1	10	22				6

Case XV—J. T.

	Separate Cold Stimuli				Double Stimuli										
					Response C–H							Response Cold			
	Med.	P. E.	No. of Cases		1st Interval		2d Interval		No. of Cases			Med.	P. E.	No. of Cases	
			3.0″+	Total	Med.	P. E.	Med.	P. E.	1st Int. 3.0″+	2d Int. 1.0″	Total			3.0″+	Total
I	1.2	.20	1	15	1.3	.35	.4	.00		1	8				
II	1.4	.20		14	1.1	.10	.4				9				
III	1.2	.25		9	1.6	.20	.6	.30	1	3	9	1.3	1.2–1.8		3
IV	1.0	1.0–1.1		2											
V	1.6	.10		8	1.5	.10	1.2	.80		6	10	2.0	1.8–2.2		2
VI	1.6	.05	1	8	1.6	.35	.8	.35		3	9	2.7	1.8–3.6	1	2
VII	1.3	.00		4	1.1	.15	.5	.50		1	7				
VIII	1.0	.10		6	1.1	.05	.4	.00		1	4				
Totals			2	66					1	15	56			1	7
	Separate Hot				Response H–C							Response Hot			
I	1.0	.05		14	1.5	.25	2.3	.65		6	6	1.3	.10		16
II	1.0	.10		15	1.3	.30	.6	.75		3	7	1.4	.05		14
III	1.3	.65	1	7	2.1	2.0–2.3	2.1	1.0–3.2		2	2	1.8			1
IV	1.0	1.0–1.0		3								1.3	1.3–1.3		2
V	1.7	.80	1	7	1.8	1.0–2.6	1.5	.6–2.4		1	2	2.0	.75	3	10
VI	1.4	.25		8	2.8	2.4–3.2	.9	.6–1.2	1	1	2	2.4	.40	1	10
VII	1.0	.15		4	1.9	.30	2.5	.30		3	4	1.3	.15		8
VIII	1.0	.00		4	1.3	1.2–1.4	.4	.4–.5			3	1.2	.25		9
Totals			2	62					1	16	26			4	70

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* Total range given with less than four cases.

TABLE 3. (Multiple Sclerosis concluded)

Case XVI—I. R.†

	Separate Cold Stimuli				Double Stimuli										
					Response C–H							Response Cold			
	Med.	P. E.	No. of Cases		1st Interval		2d Interval		No. of Cases			Med.	P. E.	No. of Cases	
			3.0″+	Total	Med.	P. E.	Med.	P. E.	1st Int. 3.0″+	2d Int. 1.0″+	Total			3.0″+	Total
I	1.1	.05		5	1.4	1.1–1.6	.5	.4–.5			3				
II	1.2	.05		4	1.5	.05	.9	.25		2	4				
III	1.0	.9–1.3*		3	1.9	1.2–2.2	.6	.4–.7			3				
IV	1.3			1											
V	1.4	1.2–1.6		3	1.5	.10	.6	.10		1	4	1.6	1.6–1.6		2
VI	2.2	1.4–2.6		3	1.9	1.1–3.7	.8	.6–.9	1		3	1.1	.9–2.4		3
VII	1.0	1.0–1.1		2	1.3	.05	.6	.10			4				
VIII	1.0			1	1.4	1.2–1.7	.9	.5–1.3		1	2	1.6			1
Totals				22					1	4	23				6
	Separate Hot				Response H–C							Response Hot			
I	1.0	.05		6	1.3	.45	1.5	.85		5	7	1.3	1.2–1.4		2
II	1.2	1.1–1.3		2	1.1	.25	.5	.00		1	5	1.4			1
III	1.0	.8–1.3		3											
IV	(No	stimuli)			1.6		.4				1				
V	1.6	1.4–1.8		2	2.2		.6				1				
VI	2.2	1.3–2.3		3											
VII	1.0	1.0–1.0		2											
VIII	1.0			1								1.6			1
Totals				19						6	14				4

Case XVII—J. B.†

	Separate Cold Stimuli				Double Stimuli										
					Response C–H							Response Cold			
	Med.	P. E.	No. of Cases		1st Interval		2d Interval		No. of Cases			Med.	P. E.	No. of Cases	
			3.0″+	Total	Med.	P. E.	Med.	P. E.	1st Int. 3.0″+	2d Int. 1.0″+	Total			3.0″+	Total
I	.9	.00		6	.9	.05	1.3	.15		6	8				
II	1.0	.05		6	1.0	.15	1.3	.55		4	7				
III	.9	.8–1.2		3	1.0	1.0–1.2	1.3	1.2–4.2		3	3				
IV	.9			1											
V	1.2	1.0–1.2		3	1.0	.00	1.3	.10		4	4	1.2			1
VI	1.1	.9–1.2		3	.9	.9–1.0	1.4	1.4–1.5		2	2	1.1	.10		4
VII	.9	.9–1.0		2	1.6	1.2–1.9	.6	.6–1.6		1	3				
VIII	.9	.9–.9		2	1.2	1.1–1.3	.8	.7–.9			2	1.2			1
Totals				26						20	29				6
	Separate Hot				Response H–C							Response Hot			
I	1.0	.10		6											
II	1.1	.05		6											
III	1.0	.8–1.4		3	1.2		2.8			1	1				
IV	1.6			1											
V	1.1	1.0–1.6		3											
VI	1.4	.9–1.6		3											
VII	1.0	1.0–1.0		2	2.1		3.2			1	1				
VIII	1.0	1.0–1.0		2											
Totals				26						2	2				

* Total range given with less than four cases.

† Patient came only once.

TABLE 4

MEDIAN REACTION-TIME FOR SEPARATE AND DOUBLE STIMULI IN PERIPHERAL NEURITIS

Case XVIII—L. R.

	Separate Cold Stimuli				Double Stimuli										
					Response C–H							Response Cold			
	Med.	P. E.	No. of Cases		1st Interval		2d Interval		No. of Cases			Med.	P. E.	No. of Cases	
			3.0″+	Total	Med.	P. E.	Med.	P. E.	1st Int. 3.0″+	2d Int. 1.0″	Total			3.0″+	Total
I	1.2	.05		11	1.4	.25	.5	.10		2	27				
II	1.2	.20		11	1.2	.05	.4	.00			24				
III	1.1			1	1.4	.05	.4	.15		1	5				
Totals				23						3	56				
	Separate Hot				Response H–C							Response Hot			
I	1.2	.05		13	1.8	.40	.6	.80		2	6	1.1			1
II	1.2	.15		9	1.8	.40	.8	.00		1	4	2.6			1
III	2.2			1											
Totals				23						3	10				2

Case XIX—M. F.†

	Separate Cold Stimuli				Double Stimuli										
					Response C–H							Response Cold			
	Med.	P. E.	No. of Cases		1st Interval		2d Interval		No. of Cases			Med.	P. E.	No. of Cases	
			3.0″+	Total	Med.	P. E.	Med.	P. E.	1st Int. 3.0″+	2d Int. 1.0″+	Total			3.0″+	Total
I	1.6	1.2–2.0*		2	1.4	.15	.5	.05		1	5				
II	1.2	1.2–2.0		3	1.5		.4				1	4.5	2.9–6.2	1	2
Totals				5						1	6			1	2
	Separate Hot				Response H–C							Response Hot			
I	1.3	1.3–1.4		2	1.4	1.3–1.6	.6	.5–.7			2				
II	1.3	1.2–1.4		2	2.0	.25	1.0	.30		6	11	1.7	1.6–4.0	1	3
Totals				4						6	13			1	3

Case XX—H. T.†

	Separate Cold Stimuli				Double Stimuli										
					Response C–H							Response Cold			
	Med.	P. E.	No. of Cases		1st Interval		2d Interval		No. of Cases			Med.	P. E.	No. of Cases	
			3.0″+	Total	Med.	P. E.	Med.	P. E.	1st Int. 3.0″+	2d Int. 1.0″+	Total			3.0″+	Total
I	1.1	.00		5	1.0	.15	.6	.05		1	4				
II	1.1	.00		4	1.0	.05	.8	.05		1	4				
III	1.1	.9–1.2		3	} 1.1	.10	.8	.05			4				
IV	(No	stimuli)													
V	1.1			1	1.1	.05	.8	.10		1	4				
VI	(No	stimuli)			1.2	.05	.8	.00		1	4				
Totals				13						4	20				
	Separate Hot				Response H–C							Response Hot			
I	.8	.10		5	1.2	.15	1.0	.30		7	12				
II	.9	.00		5	1.1	.05	.8	.25		5	12				
III	.8	.8–1.0		3	} 1.2	.20	1.0	.30		5	7				
IV	(No	stimuli)													
V	(No	stimuli)			1.4	.05	1.0	.10		1	1				
VI	1.0	1.0–1.1		2											
Totals				15						18	32				

* Totals given with less than four cases.

† Patient came only once.

TABLE 4. (Peripheral Neuritis concluded)

Case XXI—B. P.†

	Separate Cold Stimuli				Double Stimuli										
					Response C–H							Response Cold			
	Med.	P. E.	No. of Cases		1st Interval		2d Interval		No. of Cases			Med.	P. E.	No. of Cases	
			3.0″+	Total	Med.	P. E.	Med.	P. E.	1st Int. 3.0″+	2d Int. 1.0″+	Total			3.0″+	Total
I	1.8	.10		5	2.7	.30	.4	.05	4		8	2.6			1
II	1.7	.30		5	2.4	.55	.5	.15	5	1	12				
III	1.6	1.6–2.0		3	2.8		.6				1	1.8	.10		4
Totals				13					9	1	21				5
	Separate Hot				Response H–C							Response Hot			
I	1.9	.10		5								3.0	.20	3	5
II	1.6	.15		5											
III	2.1	1.5–2.6		3	2.3		1.1			1	1				
Totals				13						1	1			3	5

† Patient came only once.

TABLE 5

MEDIAN REACTION-TIME FOR SEPARATE AND DOUBLE STIMULI IN A CASE OF THALAMIC LESION

Case XXII—D. L.

	Separate Cold Stimuli				Double Stimuli										
					Response C–H							Response Cold			
	Med.	P. E.	No. of Cases		1st Interval		2d Interval		No. of Cases			Med.	P. E.	No. of Cases	
			3.0″+	Total	Med.	P. E.	Med.	P. E.	1st Int. 3.0″+	2d Int. 1.0″+	Total			3.0″+	Total
I	2.2	.25		12								2.2	.35	4	30
II	2.0	.40		11	2.6	.55	1.1	.35	4	14	17				
III	2.3	.55	1	6	2.6	.40	1.2	.20	2	3	4	2.4	.00	1	4
IV	2.4	2.3–2.6		2											
V	2.4	.45	1	7								2.6	.35	6	24
VI	2.6	.40	2	5	2.6	.65	1.1	.30	5	14	15	2.8	.15	2	4
VII	2.3	.10		4	2.2	1.8–2.9	1.0	.9–1.2		2	3	2.6	.25		5
VIII	2.5	.50	2	4	2.5	1.9–3.0	1.2	1.0–2.1	1	3	3	2.8	.70	4	11
Totals			6	51					12	36	42			17	78
	Separate Hot				Response H–C							Response Hot			
I*															
II	1.7	.10	1	12	2.0	.15	.8	.15		2	9	2.0	.35		5
III	2.6	.25	1	5	2.4	.15	1.1	.50		2	4	2.7	.15		4
IV	2.0	2.0–2.0		2											
V*															
VI	2.9	.15	2	4	2.2	.30	.6	.10	1	2	8				
VII*	2.7			1	2.0	.20	.6	.30		2	7	2.2			1
VIII	*1.7			1	3.2	3.0–3.5	.6	.6–.7	2		2	1.5			1
Totals			4	25					3	8	30				11

* In response to hot stimulus:
Region I gave 10 responses *Cold*-Med. 2.8; P. E. .40. Region VII gave 2 responses *Cold*-Med. 2.7; *range* 2.7–3.2.
Region V gave 6 responses *Cold*-Med. 3.6; P. E. .95. Region VIII gave 1 response *Cold*-Med. 3.8.

TABLE 6

PERVERSION OF RESPONSE—PERCENTAGE OF STIMULI GIVING OPPOSITE RESPONSE

	Syringomyelia												Tabes Dorsalis									
	P. K.		E. Wi.		A. C.		L. A.		J. Cl.		B. T.		G. C.		P. O.		J. Co.		L. F.		H. D.	
	C	H	C	H	C	H	C	H	C	H	C	H	C	H	C	H	C	H	C	H	C	H
Hand, R	100			50	100						Right arm 100											
L					100														17			
Forearm, R	40	35			100		50												20			
L					50														17			
Arm, R	40	50			67														17			
L	40					50												17	67			
Face, R																			40			
L									67*													
Neck																						
Leg, R													100						50			
L													100						67			
Thigh, R							50		50*										100			
L									50*										67			
Foot, R																50			33			
L				50					50*							50	33		33			
Abdomen, R					33	33	33		63*										50			
L																						
Back, R									80*						25		33		67			
L																						

	Multiple Sclerosis												Neuritis								Thal. Les.	
	E. Wa.		J. Q.		G. P.		J. T.		I. R.		J. B.		L. R.		M. F.		H. T.		B. P.		D. L.	
	C	H	C	H	C	H	C	H	C	H	C	H	C	H	C	H	C	H	C	H	C	H
Hand, R																						100
L																						
Forearm, R																						100
L																						
Arm, R																						50
L																						
Face, R																					33	
L																						
Neck																						
Leg, R																						100
L						33																
Thigh, R																						100
L																						
Foot, R																						100
L																						
Abdomen, R																						100
L																						50
Back, R																						50
L																						

* Includes some cases in which a very dubious correction was made, from .8 to 5.0 seconds later.

TABLE 7

FAILURE TO REACT—PERCENTAGE OF STIMULI GIVING NO RESPONSE

Syringomyelia

	P. K.			E. Wi.			A. C.			L. A.			J. Cl.			B. T.	
	C	H	D	C	H	D	C	H	D	C	H	D	C	H	D	C H	D
Hand, R							88		75				100	100	100	Right arm—C & H–17%	
L				100	100	94	88	80	88				100	100	100		38
Forearm, R								67	100				100	100	100		
L						13	50	100	100				100	100	100	Left arm—C & H—67%	
Arm, R								67					100	100	100		63
L							75	40	75				100	100	100		
Face, R																	
M																	
L																	
Neck							100	100									100
Leg, R																	
L																	
Thigh, R																	
L																	
Foot, R												25					
L												25					
Abdomen, R							25	25									
M																	
L																	
Back, R							100	100	100*								
M																	
L																	

Multiple Sclerosis

	E. Wa.			J. Q.			G. P.			J. T.			I. R.			J. B.		
	C	H	D	C	H	D	C	H	D	C	H	D	C	H	D	C	H	D
Hand, R																		
L																		
Forearm, R																		
L																		
Arm, R																		
L																		
Face, R																		
L																		
Neck																		
Leg, R																		
L																		
Thigh, R																		
L						13												
Foot, R																		
L																		
Abdomen																		
Back																		
R																		
M																		
L																		

C indicates cold stimuli; H, hot; and D, double stimuli.

* Areas usually stimulated gave no response; responses were obtained on shoulder blades.

TABLE 7. (concluded)

Tabes Dorsalis														
G. C.			P. O.			J. Co.			L. F.			H. D.		
C	H	D	C	H	D	C	H	D	C	H	D	C	H	D
								25						
							67							

Neuritis												Thalamic Lesion		
L. R.			M. F.			H. T.			B. P.			D. L.		
C	H	D	C	H	D	C	H	D	C	H	D	C	H	D
			33	33	14†									
													50	25
													50	

se percentages were with temperatures of 16–17° and 46–50° C.; temperatures of 18° and 40° evoked no response on the median ce of the left hand.

TABLE 9

DIMINUTION OF SENSATION—PER CENT. OF WARM RESPONSES

Region	Syringomyelia												Tabes Dorsalis									
	P. K.		E. Wi.		A. C.		L. A.		J. Cl.		B. T.		G. C.		P. O.		J. Co.		L. F.		H. D.	
	D	S	D	S	D	S	D	S	D	S	D	S	D	S	D	S	D	S	D	S	D	S
I	97	64	94	58	67	50	100	100			100	100	38	31			32	17			71	33
II	76	92	70	28	100	67	100	100			100	100	12	23			50	47			18	55
III	67	71	91	50	40	33	100	100			100	100	20				25					33
IV	100	100	50	50			100	100			100	100					75	50				
V	83	67	81	83	8		100	100			100	100	18	44			75	86			60	4
VI	94	83	100	50		33	100	100			100	100	38	33			57	71			60	5
VII	38	50	88	100	60	25	100	100			100	100					33					2
VIII	100	75	53	50	25		100	100			100	100	27				29					

DIMINUTION OF SENSATION—PER CENT. OF COOL RESPONSES

	Syringomyelia												Tabes Dorsalis									
	P. K.		E. Wi.		A. C.		L. A.		J. Cl.		B. T.		G. C.		P. O.		J. Co.		L. F.		H. D.	
	D	S	D	S	D	S	D	S	D	S	D	S	D	S	D	S	D	S	D	S	D	S
I	7												25	14								
II	10	14											14								13	
III					50								16									
IV													50									
V													50									
VI													27									
VII													14									
VIII																						

TABLE 10

INTENSIFICATION OF SENSATION

	Syringomyelia												Tabes Dorsalis									
	P. K.		E. Wi.		A. C.		L. A.		J. Cl.		B. T.		G. C.		P. O.		J. Co.		L. F.		H. D.	
	D	S	D	S	D	S	D	S	D	S	D	S	D	S	D	S	D	S	D	S	D	S
I																						
II																				1C*		
III																			1HC			
IV																						
V					1H									1C	1CH 2C	1C				1C*		
VI					2H	1C									1HC 1C				1HC	1C		
VII													1HC 1H		2CH 1HC 3H& 3C				4HC 4H	6C† 5H†	1CH 1C 2H	2C 2H
VIII													2H	1C		1C* 3H	2H 1C	1C 1H	4HC 5H	6C¶ 5H‡	1H	

Under D is shown the number of cases of reflex retraction from a double stimulus, with initials indicating the type of response; under S reflexes occurring with single stimuli—initials indicating which.

[TABLE 8 WILL BE FOUND ON PAGE 364]

Multiple Sclerosis												Neuritis								Thalamic Lesion	
E. Wa.		J. Q.		G. P.		J. T.		I. R.		J. B.		L. R.		M. F.		H. T.		B. P.		D. L.	
D	S	D	S	D	S	D	S	D	S	D	S	D	S	D	S	D	S	D	S	D	S
25	92	100	100	93	92	6				100	100	100	100	14				77	40		
6	42	100	100	90	85					100	100	100	100	19				78	80		
30	50	100	100	92	100	30	43			100	100	100	100			11	33	50	100		
25	8	100	100	100	50					100	100										
65	100	100	85	100	100	23				100	100										
58	100	100	100	100	85	23				100	100										
44		100	100	93	100	5				100	100										
15	20	100	100	92	50					100	100										

Multiple Sclerosis												Neuritis								Thalamic Lesion	
E. Wa.		J. Q.		G. P.		J. T.		I. R.		J. B.		L. R.		M. F.		H. T.		B. P.		D. L.	
D	S	D	S	D	S	D	S	D	S	D	S	D	S	D	S	D	S	D	S	D	S
					7		7							14							
						6		11						60							
						7															
						8			33												

Under D is to be found the per cent. of responses to double stimuli including any perception of the hot stimulus, which ere *warm;* under S the per cent. of warm responses to single hot stimuli.

Multiple Sclerosis												Neuritis								Thalamic Lesion	
E. Wa.		J. Q.		G. P.		J. T.		I. R.		J. B.		L. R.		M. F.		H. T.		B. P.		D. L.	
D	S	D	S	D	S	D	S	D	S	D	S	D	S	D	S	D	S	D	S	D	S
																				1C	2C
								1CH												6C	1C&1H
								2C													
								1C													
								4CH			1C										1C
		4C	1C			1H	1C				1C									1C	1C&1H

* Response was H-C or H.

¶ 3 responses were H-C, 1 H.

† 3 responses were H-C.

‡ 2 responses were H-C.

TABLE 8

CONSISTENCY OF RESPONSE

Single Stimuli	Syringomyelia			Tabes Dorsalis					Multiple Sclerosis				Neuritis Thalamic Lesion	
	P. K.	E.Wi.	J. Cl.	G. C.	P.O.	J. Co.	L. F.	H. D.	E.Wa	J. Q.	G. P.	J. T.	L. R.	D. L.
Per cent. of areas having														
Cold—2 C responses	*56*	92	*44*	*73*	96	96	*46*	100	100	100	89	100	90	92
Hot—2 H responses	*84*	*73*	100	*50*	92	96	*69*	100	100	100	96	100	100	*44*
Cold—range 1.0″	*14*	*17*		*16*	*24*		*17*	*23*	*23*		*8*	*23*		*20*
Hot—range 1.0″	*10*	*11*	6	*10*	4	8	*28*	*20*	*12*		4	*19*		*27*
Cold—range <.3″	*29*	*47*	*19*		*32*	60	*33*	*35*	65	77	50	*12*	56	*24*
Hot—range <.3″	*33*	50	69	*35*	64	*52*	*28*	*24*	*42*	73	52	69	75	*36*
Double Stimuli														
Per cent. of areas having														
2 or more responses—C–H	*40*	*48*	*17*	*28*	*38*	*45*	*8*	*3*	*20*	*48*	75	*43*	89	*35*
2 or more responses—H–C	8	18		33	20	5	*70*		25		10	13	16	20
2 or more responses—C	*18*	*13*	8	*18*	*33*	*28*	*15*	*65*	*15*	*48*	*23*	6		*48*
2 or more responses—H	*25*	3	*96*	*25*	*23*	*35*	*8*	*20*	*45*	*23*		*75*		5
C–H, 1st—range 1.0″	*19*	*35*	0	*27*	*54*	*6*	*33*	*100*	0	0	*23*	*29*	*25*	*50*
2nd—range 1.0″	25	21	25	27	*33*	28	0	*100*	*63*	*42*	*40*	24	0	*35*
C–H, 1st—range < .3″	*25*	*24*	100	91	*7*	67	*33*	*0*	50	58	*23*	47	*25*	0
2nd—range < .3″	*38*	*35*	50	*27*	*27*	*39*	67	*0*	*13*	*16*	*30*	53	70	*21*
H–C, 1st—range 1.0″	*33*	*29*		*65*	*38*	0	*25*		10		0	*40*	0	13
2nd—range 1.0″	*67*	29		*38*	*50*	*50*	18		*90*		*75*	*60*	*67*	*38*
H–C, 1st—range < .3″	*0*	43		*8*	38	50	*22*		40		0	40	33	38
2nd—range < .3″	33	43		23	50	50	68		*0*		*0*	*0*	33	*0*
C—range 1.0″	28	60	0	57	46	0	0	54	33	0	44	50		63
C—range < .3″	29	0	0	14	15	46	17	8	17	68	11	0		0
H—range 1.0″	40	0	0	40	44	21	0	50	28	13		7		100
H—range < .3″	10	100	22	50	22	29	50	13	39	50		43		0

The percentage of areas having two responses of a kind is calculated on the basis of all areas stimulated—forty for double stimuli, twenty-six for single stimuli (except in cases where for some reason an area failed to receive two stimulations).*

The percentage of areas having a given range is calculated on the basis of the number of areas giving two responses of the sort indicated.

Figures in italics indicate percentages outside of normal limits—*i. e.*, deviating from the normal average by more than two and a half times the average deviation. But C or H responses among normals were so infrequent that the time-fluctuation was not calculated.

* For single stimuli, two responses of a kind indicates 100 per cent. for that area, in general; for double stimuli, 50 per cent.

[OWING TO EXIGENCIES OF MAKE-UP, TABLES 9 AND 10 WILL BE FOUND ON PAGES 362 AND 363]

TABLE 11

COMPARISON OF THE REACTION-TIME OF COLD AND HOT IN SINGLE STIMULI

	Time of C > H		Time of H > C		Time of C = H		Time of C > H		Time of H > C		Time of C = H
	No. of Regions	Average Amount Sec.	No. of Regions	Average Amount Sec.	No. of Regions		No. of Regions	Average Amount Sec.	No. of Regions	Average Amount Sec.	No. of Regions
Syringomyelia						**Multiple Sclerosis**					
P. K.	4	.50	3	.23	1	E. Wa.	3	.23	4	.20	1
E. Wi.	3	.13	5	.44		J. Q.	2	.10	5	.22	1
A. C.	2	1.05	5	1.00	1	G. P.	2	.20	3	.13	3
L. A.	5	.48	3	.40		J. T.	4	.28	2	.10	2
J. Cl.	5	.24	1	.10	2	I. R.	3	.05	1	.20	4
B. T.	2	1.90	2	.70	1	J. B.	1	.10	7	.21	
Average	3.8	.48	3.4	.43		Average	2.8	.17	3	.17	
Tabes Dorsalis						**Neuritis**					
G. C.	6	.37	2	.45		L. R.	-		1	1.10	2
P. O.	4	.48	1	.10	3	M. F.	1	.30	1	.10	
J. Co.			6	.32	2	H. T.	4	.23			
L. F.	3	.40	5	.30		B. P.	1	.10	2	.30	
H. D.	5	.40	2	.80	1	Average	1.5	.16	1	.37	
Average	3.6	.34	3.2	.39							
Thalamic Lesion											
D. L.	3	.50	3	.40							

TABLE 12

COMPARISON OF THE REACTION-TIME OF COLD AND OF HOT RESPECTIVELY IN SINGLE AND DOUBLE STIMULI

	C & C–H	H & H–C		C & C–H	H & H–C		C & C–H	H & H–C		C & C–H	H & H–C
P. K.	+ .1	+2.1	G. C.	+6.3	+7.4	E. Wa.	+1.7	+1.0	L. R.	+ .5	+1.2
E. Wi.	+ .8	+1.4	P. O.	+2.6	+4.8	J. Q.	− .4		M. F.	+ .1	+ .8
A. C.			J. Co.	+1.6	+ .6	G. P.	+1.3	+3.1	H. T.	− .2	+1.0
L. A.	−1.8	+ .1	L. F.	+ .7	−1.8	J. T.	0	+4.3	B. P.	+2.8	
J. Cl.	− .2		H. D.	+ .4*	+2.7	I. R.	+1.9	+ .8			
B. T.	− .5*	+ .9*				J. B.	+ .7		D. L.	+ .8	+ .4
Av.	− .3	+1.1		+2.3	+2.7		+ .9	+2.3	(Neur.)	+ .8	+1.3

These figures represent the sum of the gains in time of medians (in seconds) for each individual in double stimuli over medians in single for same region. Starred figures, however, are gains of averages, so calculated because of too few cases to calculate by regions.

CHART A

SUMMARY OF DISTURBANCE IN ALL CASES

	Syringomyelia						Tabes Dorsalis				
	P. K.	E. Wi.	A. C.	L. A.	J. Cl.	B. T.	G. C.	P. O.	J. Co.	L. F.	H. D.
Failure to react		+	+++	+	++++	++			+		
Perversion	++	+	+++	++	+++	++++	+++	+	++	+++	
Type of response	++	+	++++	+++	++++	++	++++	++++	++	++	++++
Median reaction-time	+++	++	++++	++	+	++	++	+	+	+	+++
Extreme reactions	+++	+++	++++	+++	++	++	++++	+++	+	+	++++
Consistency	++++	+++	No meas.	No meas.	++	No meas.	++++	+++	++	+++	+++
Diminution of sensation	++++	++++	++	++++		++++	+++		++		++
Intensification	+		+				+	++	+	+	++
Relatively greater disturbance	$C_s H_d$	H	C	C	C	C	C	$C_s H_d$	H	C	$C_s H_d$
Degree of disturbance	++	++	++++	++	++++	+++	+++	++	+	+	+++
Disturbance general	+		+	+			+	+	+	+	+
Disturbance local		+			+	+					

	Multiple Sclerosis						Neuritis				Thalamic Lesion
	E. Wa.	J. Q.	G. P.	J. T.	I. R.	J. B.	L. R.	M. F.	H. T.	B. P.	D. L.
Failure to react		+						+			+
Perversion			+								++++
Type of response	++++	++++	+	+++	+	+		+	+	+	++
Median reaction-time	+	+	+	+	+	+		++	+	++	++
Extreme reactions	++	++	+	++	+	++		+++	++	+++	++++
Consistency	+++	++	+	++	No meas.	No meas.	+	No meas.	No meas.	No meas.	++++
Diminution of sensation	++	++++	++++	+		++++	++++	+	+	++++	
Intensification		+	+	+	+	+					++
Relatively greater disturbance	C = H	H	H	C	C = H	H	–	C	C?	?	H
Degree of disturbance	+	+	+	++	+	+	–	+++	?	?	++++
Disturbance general	+	+		+		+			?	?	
Disturbance local			+		+			+			+

In each aspect of the experiment, as designated on the left, the relative degree of disturbance is indicated for each subject by the number of plus signs—except in the last two, where the sign signifies whether the disturbance was general or local in character.

That there is in most of these cases a departure from the normal reaction is evident; and that the double stimulation shows a disturbance not always evident with separate stimuli is also clear. Perhaps the nature of the disturbance will be more quickly grasped by the summary in Chart A, in which the estimated degree of disturbance shown by each subject in the various aspects of the test is indicated by plus signs.

The chart is also a convenient reference table for comparing the various syndromes. There are a few salient points to be noted:

1. Failures are by far the most frequent in syringomyelia.
2. Perversions are commonest in syringomyelia; but are frequent also in tabes, and in the case of thalamic lesion.
3. In type of response neuritis deviates least from normal—but these cases for the most part showed no clear-cut disturbance. Other syndromes do not differ significantly in this respect one from another.
4. In reaction-time multiple sclerosis is nearest to normal. If reference is made to Tables 1–5 it will be seen that these cases are generally prompt in reaction to separate stimuli; and that they generally show a retardation, chiefly in the second interval of double responses.
5. Extreme reactions in the second interval of double responses only are characteristic of multiple sclerosis. Except in multiple sclerosis extreme reactions are also frequent in single responses to double stimuli.
6. Diminution of intensity of sensation is least and intensification is most in tabes dorsalis.
7. Cold is more likely to show a greater disturbance than hot in syringomyelia; hot more than cold in multiple sclerosis.
8. All factors considered, multiple sclerosis shows the least degree of disturbance.
9. The disturbance is unlikely to be local in tabes, most likely to be local in syringomyelia.

Summarizing, one may say that under the conditions of the experiment—for these subjects at least—frequency of failures and of perversions, generally lengthened reaction-times, diminution of sensation, and tendencies toward marked local disturbance are indicative of syringomyelia. Perversions without failures; somewhat lengthened reaction-time—especially in double stimuli; tendencies toward intensification; and general, rather than local, character of disturbance suggest tabes. A generally normal or nearly normal reaction—except for a lengthened interval between the perception of the two stimuli—is characteristic of multiple sclerosis. The lesion of the brain stem is distinguished from those of the cord by the unilateral character of the disturbance, and by the clear-cut selection of one sort of sensation as evidenced by the responses. Peripheral disturbances, such as traumatic neuritis, are not adequately shown by this method of testing.

There are a few points which may be of general interest to the theory of temperature sensations. One is the relation of the order of response in double stimuli to the length of reaction of cold and hot separately. It will be remembered that with *normals*, while the time for cold and hot was about the same, the majority of responses to the double stimulus were in the order *cold-hot* and that these responses were slightly more prompt and less variable, in both intervals, than those in the order *hot-cold*;[7] that the responses of hot followed quite regularly upon those of cold except on legs and feet, where it was somewhat delayed. In testing *abnormals* we have an opportunity to see what happens when the times of hot and cold are more variable.

Table 11 gives a summary of the relation of cold and hot on each region, for the various subjects. We find, that in spite of greater variability there is, generally considered, the same overlapping in the times of cold and hot that was found among normals. There are four subjects, however, in whom is shown a fairly distinct tendency of hot to be slower—E. Wi., J. Co., J. Q. and J. B.; and there are six who show a somewhat slower time for cold—P. K., J. Cl., G. C., P. O., H. D. and J. T. Of the cases in whom cold is quicker, we find, by comparing the frequency of different types of response to double stimuli (Tables 1–5), that C–H responses exceed H–C in all cases, and C exceed H in all but one case; that C–H is the most frequent type in all but one case, in whom C has the highest frequency. On the other hand, among those

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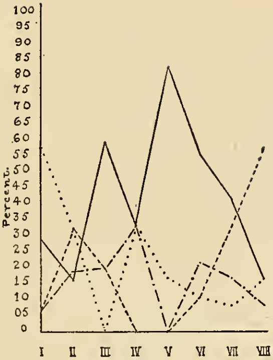
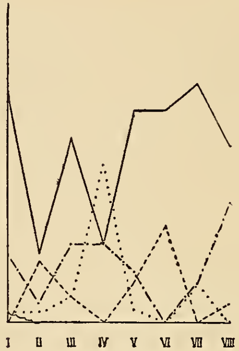
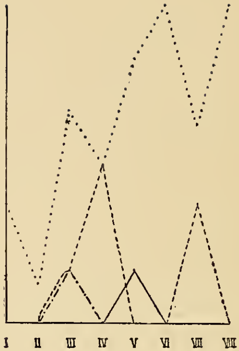
[7] Neurological Bulletin, loc. cit., Table 12, p. 156



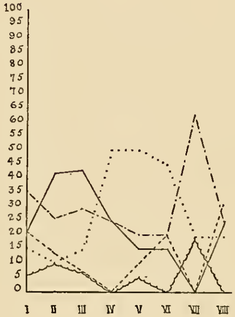
P.K.



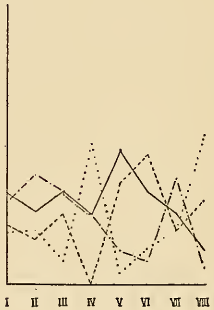
E.Wi.



A.C.



G. C.



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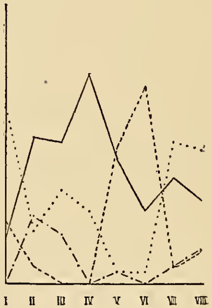
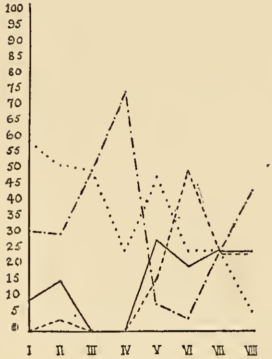
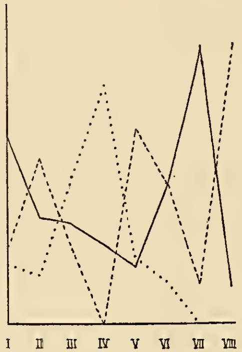


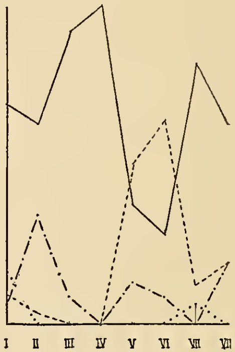
J. Co.



E.Wa.



J. Q.



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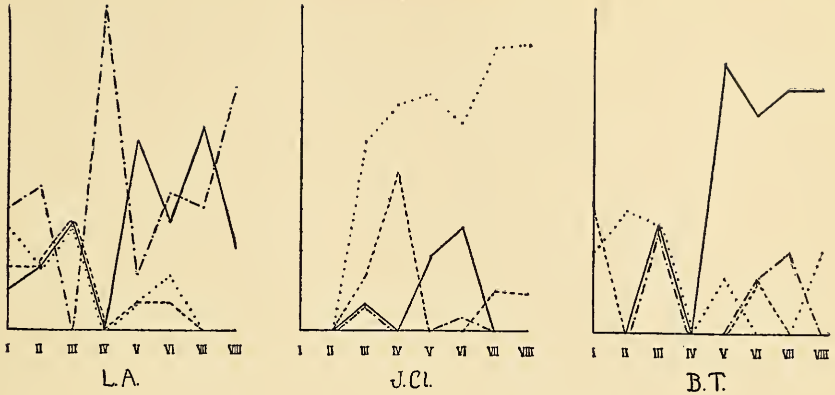


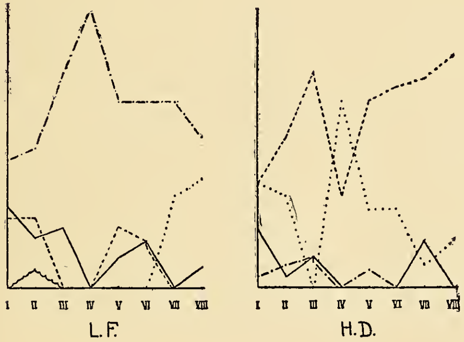


FIG. 1. (TOP) DISTRIBUTION OF TYPES OF RESPONSE IN SYRINGOMYELIA

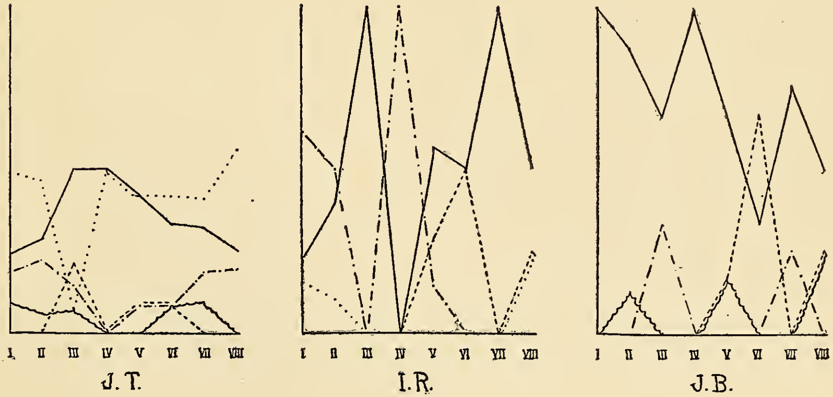
— C-H -.- H-C --- C .... H ⁓ Duobtful

FIG. 2. (CENTRE) DISTRIBUTION OF TYPES OF RESPONSE IN TABES DORSALIS

— C-H -.- H-C --- C .... H ⁓ Doubtful

FIG. 3. (LOWER) DISTRIBUTION OF TYPES OF RESPONSE IN MULTIPLE SCLEROSIS

— C-H -.- H-C --- C .... H ⁓ Doubtful



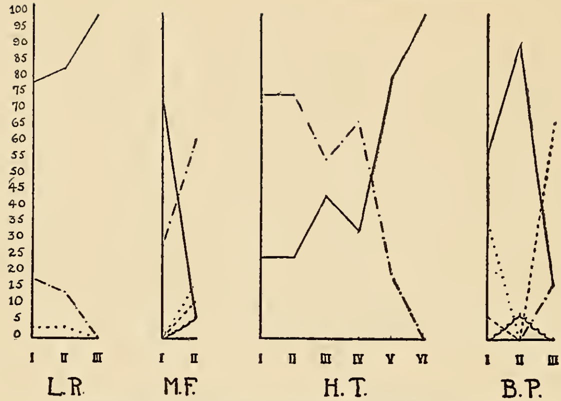


FIG. 4. DISTRIBUTION OF TYPES OF RESPONSE IN PERIPHERAL NEURITIS
— C-H -.- H-C —— C .... H ‿ Doubtful

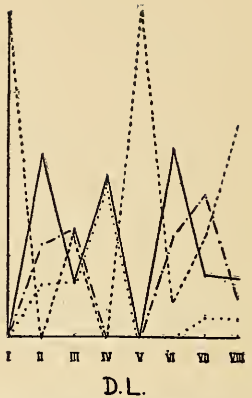


FIG. 5. DISTRIBUTION OF TYPES OF RESPONSE IN A CAPSULOTHALAMIC LESION

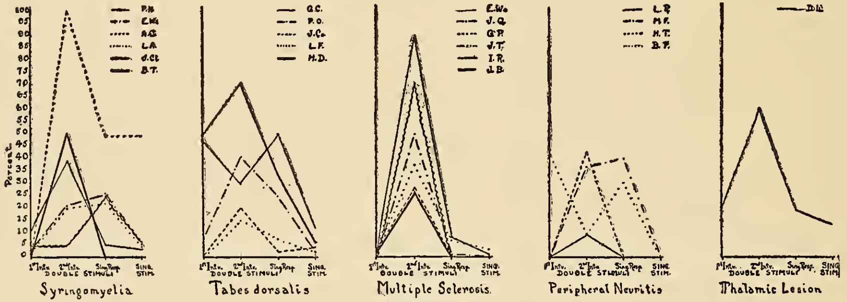


FIG. 6. EXTREME REACTIONS
A graph showing manner in which extreme reactions were distributed among the different types of response

who show a slightly slower reaction to cold, H–C responses exceed C–H in only one case, though H are more frequent than C in four cases; and the most frequent type in three cases is C–H or C, although in three others, it is H or H–C. Thus, the frequency of the type of response does not invariably depend on the length of the reaction to separate stimuli. Indeed, as results show, the entire character of the disturbance may be greater for hot in double stimuli, in spite of a slower cold reaction. The opposite, however, does not occur.

Another point of interest is the effect of double stimulation upon the length of reaction-time. If the gains in the medians for the double stimulus over the single, for each region, are added algebraically, for each subject, we get a rough measure of the effect of the double stimulus. This is shown in Table 12. The same sort of measure for normals can be easily computed from Table 12 of the former article. It will show that while the reaction to cold remained practically the same in C-H responses as in single stimuli (sum of the gains, total, equals —.1 second), the reaction to H in H–C responses was somewhat lengthened (sum of gains, +2.9 seconds).

The tendency of the clinical subjects is in general to increase the time for either response in double stimuli. The increase is greater for hot than for cold in twelve cases, and less in five—the remaining five patients having too few H–C responses to admit of comparison. The five cases showing less increase for hot than for cold are not, however, of those who react more slowly to cold. There are four cases that show a decrease in time of cold in double stimuli; in only one, however, is the difference very considerable. There is but one case showing a decrease in time of hot; this is the subject who reacted to almost all stimuli—single and double—by H–C. The tendency is, then—as in normals—for double stimuli to increase the reaction-time of hot more than of cold.

This leads us to the third point of general interest: that the C-H type of response indicates the least abnormality of response. It was noted even among normals that H-C responses were more irregular when they occurred than C-H, but it was assumed that this was because of the fewer number of cases of this type. Among the subjects of the present inquiry the same phenomenon appears. A slighter degree of disturbance for C-H than for H-C responses is shown by every subject—whether cold or hot is more disturbed—in the time of either first or second interval, or the proportion of extreme reactions, or in all three ways—except in the case of D. L., in whom H-C responses, although less frequent, are shorter in both intervals and less apt to be extreme. Single responses to double stimuli, are in general, longer and more apt to be extreme than double responses; that is, they indicate the greatest divergence from the normal, except in multiple sclerosis, where the only significant divergence is the lengthening of the second interval.

Here it may be observed that this seems to imply that the process of dissociation in multiple sclerosis is of a different character from that in syringomyelia or tabes in that, instead of retarding the initial perception when the double stimulus is given, the tendency is to lengthen, if not to inhibit, the perception of the second. Whereas in tabes, the prolongation of the initial perception is quite marked (*cf.* Table 12). In syringomyelia, however, the reaction is slow even for separate stimuli, and the curious result is that in three subjects, C–H responses are quicker than responses to cold separately—another proof of the slighter disturbance indicated by the C–H type of response. It may be that this retardation of the perception of the second stimulus is dependent on the degree of disturbance rather than characteristic of a particular syndrome: H. T., a neuritis case with scarcely any disturbance, and J. Cl., a syringomyelia case in whom the disturbance is quite limited, show the same general tendency. Nevertheless, since other subjects in whom the disturbance is slight do not show it, and since all the cases of multiple sclerosis tested—even those in whom the dorsal nerve roots had been severed—do show it, it seems fair to regard it as characteristic of multiple sclerosis.

To return to the points of theoretical interest. The three tendencies mentioned—that, even when cold is separately more delayed, hot is less apt to predominate in double stimuli; that in general, the perception of hot in double stimuli is more increased in time than cold, and that the exceptions to this are not the cases in which cold is separately slower; that C–H responses are nearest to normal whichever sensation is more disturbed—suggest that the double stimulation acts in a way which is not merely the additive effect of the two stimuli, but which indicates some relation between the two tending to delay the perception of hot.

The theory that best explains these facts, is, in my opinion, that which regards the perception of hot as a fusion of impulses from cold and warm spots. On this hypothesis, the double stimulation requires the cold spots to do double duty, so to speak, and naturally the fulfillment of their secondary function is delayed. This hypothesis also explains most easily the cases of perversion—at least those which do not occur with complete consistency. If cold takes part in the perception of hot, then it is not difficult to imagine the fusion disturbed and cold perceived as hot or *vice versa*. The tendencies are, however, not sufficiently unequivocal to make this interpretation of more than doubtful correctness—and there are two cases whose reaction is difficult, though not impossible, to explain on this assumption. These are J. Cl. and J. T., who show a normal reaction to cold and hot separately, somewhat quicker, however, for hot, but a considerable interference with the reaction to cold in double stimuli. It is difficult to see how the reaction to hot can remain intact, on our hypothesis, when cold is disturbed. It is necessary to assume that the cold, in exercising its second function, loses its identity in its first function because the second reaction comes so quickly. The number of instances for which both of these subjects started to say "cold" but said "hot" instead, makes this explanation not improbable.

CONCLUSIONS

1. Patients suffering from syringomyelia, tabes dorsalis, multiple sclerosis, and the type of thalamic lesion presented in this study, generally show some disturbance in reactions to double thermal stimuli, whether or not evinced by ordinary clinical tests. The temperature test, as herein presented, is not, however, well adapted to the investigation of peripheral disturbances.

2. There is a larger proportion than normal of single responses to double stimuli.

3. There is more than a normal tendency, in many cases, to call temperatures of 41° to 44°C. or higher, *warm* instead of the usual hot.

4. The disturbance is generally shown by an increased reaction-time.

5. The variability in time is greater than normal: the range of medians for different regions being greater and the consistency of single areas less.

6. Reactions to double stimuli are in general more disturbed than reactions to single; and H-C responses more than C-H.

7. C-H responses indicate the nearest approach to the normal reaction; they are the most frequent type in the least disturbed cases (with one exception); and they are nearest to normal in time for each individual.

8. The type of response to double stimuli, and the character of disturbance, cannot be predicted from the response to single stimuli.

9. The differences between the different syndromes are sufficiently distinct to be suggestive, although there is some overlapping. Multiple sclerosis has a particularly clear-cut characteristic—a dissociation of the perception of *doubleness*.

10. The double stimulation acts in such a way as to delay the perception of hot more often than that of cold.

EXPLANATION OF TABLES

Tables 1–5 show the median reaction-times of each patient in the five syndromes studied, both for separate and double stimuli, and for each type of response; the P. E., the number of extreme reactions, and the total number of responses for each region.

Tables 6 and 7 present the evidences of gross disturbance—the percentages of stimuli that gave failures and perverted responses.

Table 8 gives the percentages of consistency in type of response and in time, as is explained above. A glance at the italicized figures shows that the inconsistency was considerable among these subjects.

Table 9 shows the percentages of responses that were *warm* and *cool*, instead of *hot* and *cold*, in both separate and double stimuli. This is quite striking in view of the fact that seven per cent. of the total was the highest percentage reached by normals.

Table 10 gives the number of cases for each region of each subject in which an intensification of the sensation might be assumed by the reflex accompaniment. This is of interest in comparing the different subjects and syndromes, although no standard can be offered for reference.

Table 11 presents a comparison of the time of cold and hot responses in single stimuli. It gives for each subject the number of regions on which *cold* gave a longer time than *hot* with the average difference in time; the number of regions and the average time difference where *hot* was longer than cold; and the number of regions on which the time for cold and hot was the same.

Table 12 is a comparison of reaction-times of cold in single and double stimuli and similarly of hot.

Figures 1–5 present graphically the manner in which responses to double stimuli were distributed among the different types of response.

Figure 6. Figure 6 is a graph showing the distribution of extreme reactions among the different types of response in the various syndromes.

NEUROLOGICAL BULLETIN

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THE BROWN-SÉQUARD SYNDROME

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When one encounters the intensely interesting condition known as the Brown-Séquard Syndrome, he at first appreciates the fact that he is dealing with the symptomatic expression of a lesion of the cord for the most part unilateral. In my own experience I do not recall having seen a single case in which there was not clinical evidence of injury to more than one-half of the cord, and the syndrome referred to above was complicated by certain other signs of bilateral involvement. Trauma, resulting either from external causes, such as stab and bullet wounds, or from lesions intrinsic to the cord substances or of the meninges, very rarely produces the diagrammatic clinical representation as originally described by Brown-Séquard. Syringomyelia, neoplasms of the cord substance and of the meninges, multiple sclerosis, and syphilitic meningitis are some of the conditions in which this picture is sometimes found.

I am presenting this case today because it demonstrates the Brown-Séquard Syndrome plus signs of bilateral disturbance. I am not convinced as to the diagnosis, but I believe that there is a fair chance that surgical interference may prove of benefit.

Patient: W. W.; Case No. 22287; Male; Swede; married; age 34; occupation—stationary engineer; Came to the Vanderbilt Clinic on August 14, 1918.

HISTORY. *Present Illness.*—He complains of lameness in the left leg and shooting pains in the right leg. He has suffered from this condition over a period of five years. The onset was gradual and he states he has become progressively worse.

At first he noticed unusually severe pains in the right knee which radiated down the back of the leg to the ankle; these lasted four months. He then observed a weakness in the left leg. This weakness has progressed steadily until he has become decidedly lame. The pain in the right leg has become constantly annoying, day and night, especially when he sits or lies down. When he walks the pain is decidedly less. At times it is shooting in character. While walking relieves his pain, it causes great fatigue in the left leg; after walking a few blocks, he has to drag it.

Recently he has had urgent and frequent micturition but no incontinence. The bowels are constipated; otherwise the patient feels quite well.

Previous History.—The patient formerly had severe headaches covering a period of thirteen years. These headaches occurred three or four times a week. They were, usually, relieved at once by headache powders. Recently, however, his headaches have become much less frequent, averaging one in two weeks.

He thinks he had a chancre thirteen years ago, but had no rash or throat trouble. He dislocated his knee four years ago and made an uneventful recovery.

He states that a large ulcer appeared on the back of his right leg when his pain began. It was painless and healed in one month. It reappeared three months ago but healed in ten weeks.

Family History.—Negative.

EXAMINATION

VOLUNTARY MOTOR SYSTEM. *Abnormal Attitudes and Deformities.*—None are noted except in walking.

Gait.—The left leg is spastic and is manipulated somewhat in the hemiplegic fashion.

Coördination.—All of the tests for coördination, of the equilibratory and non-equilibratory types, are normally performed.

Skilled Acts.—Performed well.

Reflexes.—(0 = Absent; 1 = Slight; 2 = Active; 3 = Very Active; 4 = With transient clonus; 5 = With permanent clonus.)

(a) *Deep:*	*Right*	*Left*
Jaw	1	1
Supinator	2	2
Bicipital	2	2
Tricipital	2	2
Patellar	3	4
Suprapatellar	3	4
Achilles	3	5
(b) *Superficial:*		
Cilio-spinal	1	1
Upper lateral abdominal	0	0
Lower lateral abdominal	0	0
Cremasteric	0	0
Plantar flexion	0	0
Babinski	1	1

Abnormal Involuntary Movements. — No tremor can be detected in any part of the body. There are no fibrillary twitchings.

Muscle Strength.—The muscle groups of the left leg are weaker than those of the right; the right are normal. Otherwise, muscle strength is normal throughout the body.

Muscle Status.—Volume is normal. There is slight atrophy of the left leg compared with the right, but not more than one would expect from disuse. Consistency is normal. Tonus is increased in the left lower extremity, otherwise normal. Electrical reactions were not tried.

Abnormal Associated Movements.—None detected.

NERVE STATUS.—Not investigated.

GENERAL SENSORY.—Above a line drawn around the body at a level two inches above the umbilicus, all forms of sensation are normal. Below this line marked defects are found. They are, for the most part, dissociated, but not entirely so. For example, *vibration* sense is entirely lost in both lower extremities and is diminished markedly over the right and left iliac crests. *Position* and *pressure* sense are entirely preserved on both sides. The *temperature* sense is very much involved in the right lower extremity; this loss extends upward to the line two inches above the umbilicus. On the right side there is thermesthesia in certain areas and only diminution in others. In a general sense cold is better detected than heat. On the left side temperature sense is normal except for an area of hypesthesia two inches below the line referred to above. The upper level is at a point where sensibility is sharply demarcated. But after careful observation, I am unable to determine any zone of hyperesthesia. The *pain* sense is likewise abnormal. On the left there is no diminution; on the right there is marked defect up to the level referred to. There is analgesia in some parts and hypalgesia in others.

During the tests for pain and temperature when they were purposely made severe in the analgesic and anesthetic areas, the patient constantly executed defense movements with the right lower extremity even when he was unable to name the stimulus. At times the stimulus was named after a long interval between applications.

Touch is the least affected. This is accurately felt by the patient on both sides but the left is slightly increased over the right in certain areas.

The observations made in the touch, pain, and thermal tests are exactly the same posteriorly as anteriorly and extend to the same level.

CRANIAL NERVES AND HEAD.—*Olfactory and Nose.*—Normal right and left.

Optic and Oculomotor Apparatus.—The eye movements are normal. There is no strabismus or ptosis. There are a few transitory nystagmoid movements which cannot be considered pathological. The pupils are equal, regular, central, and react to light and accommodation. The fundi are negative.

There is possibly a very slight temporal pallor of the discs, but it is not sufficient to be considered pathological.

Acoustic and Ear.—The patient apparently hears normally.

Trigeminus and Mouth.—The jaw movements are normal.

Facial and Face.—Normal.

Glossopharyngeus, Vagus, Pharynx, and Larynx.—Swallowing is normal. The pharynx is of normal appearance. The palate rises equally right and left.

Hypoglossus and Tongue. — The tongue movements are normal.

MENTAL STATUS.—The patient is normal. He states that formerly he was very nervous but not so recently.

SYSTEMIC.—The *tegumentary* status is normal except for a large nevus over the middorsal region. *Glandular* status is normal. *Cardiovascular, respiratory, and gastro-intestinal status:* normal. *Sexual* status is normal. Urgency and frequency of micturition. *Skeletal* status is normal except that the patient is quite tender to percussion over the middorsal spines.

LABORATORY TESTS.—*Wassermann* of the blood is said to have been repeatedly negative. (The patient has had iodide of potassium for many months, and other antiluetic remedies, without avail.) The *spinal fluid* was negative in every respect (Dr. Jagle). *X-ray of the spine* by Dr. Le Wald is said to have been negative.

SUMMARY

The pathological findings are:

1. Bilateral pyramidal tract irritation, the left side being much more markedly involved than the right.
2. Bilateral loss of vibration sense, with preservation of position and pressure sense below the level of the seventh dorsal.
3. Loss of abdominal and cremasteric reflexes on both sides.
4. A partially dissociated anesthesia to temperature and pain on the right, with very slight involvement on the left in the abdominal segment. Touch is preserved for the most part, but the right is slightly deficient.
5. The patient suffers severe shooting pain in the right lower extremity. He is tender to percussion in the middorsal region.

In brief, then, we are dealing with a lesion of the spinal cord, located chiefly on the left side, which has been progressive over a period of five years. This lesion has apparently selected the pyramidal tracts of both sides, the posterior columns to a slight extent, and those spino-thalamic fibers decussating from the left side.

The differential diagnosis lies between syphilis of the cord or meninges, multiple sclerosis, syringomyelia, and neoplasm.

I do not believe that syphilis is an etiological factor in this case. Although the patient admits having had some venereal lesion several years ago, several blood Wassermann reactions have been negative and his spinal fluid has been proven negative. Furthermore, he has had therapeutic tests in long continued doses of antispecific remedies with no relief. He presents no other signs of neurosyphilis and I doubt whether a gumma or localized meningeal patch could produce the above picture without manifesting other clinical signs of neurosyphilis after five years.

With regard to multiple sclerosis, such a clinical picture is not often seen in this disease, but from the pathological standpoint, there is no reason why the findings reported above could not be produced by a localized sclerotic patch. But clinically we have insufficient evidence to believe that multiple sclerosis is present. The fundi are normal in my opinion, and there is no nystagmus. There is no tremor or ataxia. The deep reflexes of the upper extremities are not increased. On the contrary, they are quite normal and equal on both sides. There have been no remissions such as one finds so frequently in multiple sclerosis. Although one of the cardinal signs of multiple sclerosis is present here, namely, loss of the abdominal and epigastric reflexes, this phenomenon is easily explained on other grounds. In five years I would also expect a much greater and more extensive involvement if multiple sclerosis were present.

Syringomyelia must have thorough consideration in this case. The bizarre representations that may be seen in this disease are, of course, well known. Here we have a sensory dissociation that is syringomyelic in type. The motor symptoms conform to what one might expect in a middorsal disturbance of this sort. Atrophy, due to anterior horn involvement, is generally present in some period of the progression of syringomyelia. There is none in this instance except such as one might expect from disuse. In a general sense, the history is against this possibility, although we frequently meet with cases in which the lesion is quite well localized for a period of years. We must not overlook the point mentioned by the patient regarding a large painless ulcer that has appeared twice on the right leg. This is not apparent now, but it is of value in determining a trophic disturbance, which is common alike to syringomyelia and spinal cord neoplasms.

If it is within the range of possibility, we must afford some relief to this patient's suffering. No medicinal agents are of any value in this regard. Therefore, if surgical exploration should give promise of at least temporary comfort, I am strongly in favor of it. I believe that the evidence is in favor of a spinal cord tumor. If found, it would probably be intramedullary and decompression would be indicated, at least. My reasons for thinking that we may have to deal with a neoplasm are as follows:

1. The patient suffered severe pain in the right leg for four months previous to the onset of the motor weakness of the leg.
2. The lesion has been clearly localized for five years probably, but the symptoms have increased in intensity.
3. The shooting pain which persists in the right leg is not often observed in syringomyelia and is commonly found in neoplasm.
4. The patient is markedly tender to percussion over the middorsal spinous processes.
5. The duration of the disease is not against the possibilities of neoplasm as is well known.

If treated in the usual manner, the patient must be regarded as having a poor prognosis. Inasmuch as there is a possibility that an intramedullary tumor may be responsible for the distressing condition, I advocate a fairly extensive laminectomy, bearing in mind the fact that the seventh segment is apparently the level of the lesion. Surgical exploration can result in no harm, and it may lead to a considerable degree of relief. I am recommending the patient to the services of Doctors Tilney and Lambert at the Presbyterian Hospital for observation, with this hope in mind.

SENILE AND PRESENILE PSYCHOSES*

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The normal involution period which comes at the end of the fourth decade of life is usually followed by normal senescence which comes to all, sooner or later. The pathological changes that occur in the nervous system during the period of normal senescence and the resulting physiological changes are well known.

The individuals reaching senescence are expected to show slight lapses of memory, poor perception, failure of attention, some irritability, and somewhat blunted judgment. They are apt to speak of events of their childhood, laying greater stress on these than they do on the occurrences of every day life to which they show little attention.

The brain of such an individual at autopsy usually shows some reduction in weight, the convolutions are well rounded out and the sulci and fissures deeper than normal. Furthermore, atrophy is quite evident, and the spinal fluid is increased in amount. Microscopically, it shows a pia which is thickened, and that thickness is essentially due to increase in fibroblasts with an occasional lymphoid cell and rarely a mast cell. The neuroglia tissue is usually increased in amount; there is a distinct loss in ganglion cells; and the other cells show distinctly chronic degenerative processes. Not infrequently, an associating arteriosclerotic condition is present.

The presenile and senile psychoses, or abnormal behavior occurring in the prematurely aged and in the aged individuals, are the expressions of the qualitative and quantitative alterations from the expected behavior of individuals in the senescent period. As expressed by Charles I. Lambert (1) "When physiological involution anticipates in time, or exceeds in direction, extent, and severity, normal senescence, if it involves essentially the parenchyma, the various senile and presenile dementias are the result."

Cases of senile psychoses show a well-known picture. They usually manifest loss of memory for recent events and lack of ability to recognize objects. A tendency to reminiscence is quite marked, they become quite egotistic, not infrequently they become headstrong, peevish, and, when completely disoriented, a source of great difficulty to others. Eventually they develop a tendency to wander about the streets, conduct themselves with violence, and may even resort to criminal acts. Not infrequently they exhibit delusions of a persecutory nature. Because of the exceeding difficulty they offer to those who care for them, and, not infrequently, because of some antisocial act, these individuals are committed to hospitals for the insane.

That this clinical picture should be occasioned by distinctly pathological conditions was first suggested by Blocq and Marinesco (2) in 1892 who found in the brain of an aged epileptic certain amorphous structures. In 1898, Redlich (3) found in the brains of patients who showed clinical evidence of memory defects, mental confusion, amnesia, and aprexia, certain structures which he called miliary plaques. In 1904, Alzheimer (4) found miliary plaques in the brains of seniles. In 1906 (5), he described certain neurofibrillar changes associated with senile plaques in the brain of an individual who had aged prematurely. He attributed the presence of those plaques to the deposition of certain chemical substances resulting from faulty metabolism of the nervous elements. In 1907 Fischer (6), in studying a series of brains from old people, found the characteristic plaques only in those brains coming from senile dements, and he established a causal relationship between the presence of the senile plaques and the abnormal behavior of the senile psychotic individual. In 1909 Huebner (7), studying a similar series of senile brains, concluded that these miliary plaques were not characteristic of any special psychosis. Since then, equally competent observers have taken one or the other of these opposite stands. Barrett (8), Simchowicz (9) and Alzheimer (10) could see a causal relationship between the two; while Tiffany (11) and Fuller (12) would not accept the presence of senile plaques as characteristic of any special form of mental disease.

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*Address delivered at the Clinical Conference of the Department of Neurology of Columbia University, July 22, 1918.

TABLE I

TWELVE CASES CLINICALLY DIAGNOSED AS SENILE PSYCHOSIS. AVERAGE AGE 74.6 YEARS

Autopsy No.	*Name*	*Age*	*Clinical Diagnosis*	*Pathological Findings*
1657	H. C.	86	Senile psychosis Arteriosclerosis Chronic endocarditis	Brain weight, 1,265 gms. Moderate cerebral arteriosclerosis; cerebral atrophy; numerous senile plaques; moderate fibrillar thickening; chronic diffuse nephritis; and chronic endocarditis.
1664	M. R.	70	Senile psychosis Chronic myocarditis	Moderate cerebral atrophy; many senile plaques; moderate neurofibrillar alterations; chronic diffuse nephritis; chronic myocarditis.
1667	C. D. B.	74	Senile psychosis Myocarditis	Brain weight, 1,270 gms. Moderate cerebral atrophy; many senile plaques; neurofibrillar changes; chronic diffuse nephritis; myocarditis.
1666	C. L.	77	Senile psychosis Chronic myocarditis	Brain weight, 1,235 gms. Cerebral atrophy; many senile plaques and neurofibrillar alteration; myocarditis; chronic diffuse nephritis.
1671	K. L.	80	Senile psychosis	Brain weight, 1,275 gms. Cerebral atrophy and arteriosclerosis; many senile plaques and neurofibrillar changes; chronic vascular nephritis.
1694	A. V.	82	Senile psychosis (with a paranoid trend)	Brain weight, 1,325 gms. Moderate cerebral atrophy; cerebral arteriosclerosis; no plaques; no neurofibrillar changes; chronic axonal degeneration; chronic diffuse nephritis.
1702	J. O.	65	Senile psychosis (with a paranoid trend)	Brain weight, 1,325 gms. Moderate cerebral atrophy; cerebral arteriosclerosis; vascular nephritis.
1708	W. F.	74	Senile psychosis	Brain weight, 1,210 gms. Moderate cerebral atrophy; no plaques; moderate neurofibrillar changes; satellitosi; vascular nephritis; pulmonary infarct; chronic valvular endocarditis.
1709	L. S.	82	Senile psychosis	Brain weight, 1,075 gms. Moderate cerebral atrophy and arteriosclerosis; no plaques; many neurofibrillar changes; diffuse nephritis.
1714	F. B. T.	81	Senile psychosis	Cerebral atrophy; many senile plaques and nerofibrillar changes; chronic diffuse nephritis; brain weight, 1,186 gms.
1735	F. L. M.	65	Senile psychosis	Brain weight, 1,250 gms. Cerebral atrophy and arteriosclerosis; plaques and neurofibrillar changes; diffuse nephritis.
1736	F. F.	60	Senile psychosis Arteriosclerosis	Brain weight, 1,250 gms. Moderate cerebral atrophy and cerebral arteriosclerosis; no plaques; marked neurofibrillar changes; chronic diffuse nephritis.

Ten out of twelve cases showed plaques or neurofibrillar changes. The other two cases showed a paranoid trend clinically; and cerebral arteriosclerosis as the predominative pathological condition.

TABLE II

TEN CASES CLINICALLY DIAGNOSED AS DEMENTIA PRECOX. AVERAGE AGE 69.3 YEARS

Autopsy No.	*Name*	*Age*	*Clinical Diagnosis*	*Pathological Findings*
1668	A. S.	75	Dementia precox Carcinoma of liver Chronic nephritis	Brain weight, 1,060 gms. Moderate cerebral atrophy; loss of nerve cells; neuroglia increase; chronic nephritis; carcinoma of bile ducts.
1681	C. L.	67	Dementia precox Carcinoma of breast	Brain weight, 1,365 gms. Moderate cerebral arteriosclerosis; moderate fibrillar thickening; diminished number of ganglion cells; increase in neuroglia tissue; carcinoma of breast; chronic diffuse nephritis.
1684	T. W.	62	Dementia precox	Brain weight, 1,175 gms. Moderate cerebral atrophy; an occasional, doubtful, thickened nerve fibre.
1698	M. C.	77	Dementia precox	Brain weight, 1,150 gms. Moderate cerebral atrophy; no neurofibrillar changes; carcinoma of stomach; chronic nephritis.
1700	J. S.	66	Dementia precox	Brain weight, 1,375 gms. Satellitosis; chronic axonal changes; broncho-pneumonia; chronic nephritis.
1704	S. R.	63	Dementia precox Pulmonary tuberculosis	Brain weight, 1,125 gms. Moderate cerebral atrophy; pulmonary tuberculosis; chronic diffuse nephritis.
1713	T. J.	73	Dementia precox Paranoid type Epithelioma of forehead	Brain weight, 1,300 gms. Satellitosis; loss of nerve cells; bilateral broncho-pneumonia; epithelioma; chronic nephritis.
1716	S. C.	64	Dementia precox	Brain weight, 1,575 gms. Incarcerated inguinal hernia.
1720	P. R.	68	Dementia precox Paranoid type	Brain weight, 1,375 gms. Subacute nephritis; lobar pneumonia.
1730	L. L.	78	Dementia precox	Brain weight, 1,090 gms. Moderate cerebral atrophy; chronic diffuse nephritis; myocarditis.

Only one case (1681) showed moderate fibrillar thickening, but no plaques and no nerve cell changes; another case (1,684) showed an occasional doubtful thickened fiber, but no plaques nor cell changes. The others showed neither plaques nor neurofibrillar changes.

Out of a series of thirty cases studied by the author, twelve were diagnosed clinically as senile psychoses, the average age being 74.6; sixteen control cases, of which ten were dementia precox cases averaging 69.3 years of age; and six cerebral arteriosclerosis cases averaging 67.8 years of age. There was also one case of the depressive type of dementia precox, age 53, who clinically showed many symptoms of senile dementia, such as poor recent, but good remote, memory, misidentifying people, poor orientation, and showing periods of delusion; and one case of Alzheimer's presenile psychosis.

Table I shows twelve cases clinically diagnosed as senile psychoses; ten showed miliary plaques or neurofibrillar changes or both. Table II shows ten cases of clinically diagnosed dementia precox cases; only one showed moderate fibrillar thickening, no nerve cell changes, no plaques. The second case showed an occasional doubtfully thickened nerve fiber but no plaques and no nerve cell changes. Table III shows six cases of clinically diagnosed cerebral arteriosclerosis; two cases showed numerous plaques and marked nerufibrillar changes besides the arteriosclerosis. One showed slight thickening of the fibers but no plaques and no cell changes.

It would, therefore, seem logical to deduce a causal relationship between the abnormal behavior of senile psychotic individuals and the neurofibrillar alterations and miliary plaque formations in their brains. The presence of similar changes in two out of six brains of arteriosclerotic cases would only tend to prove the fact that not infrequently the changes in the vascular system of the brain go hand in hand with the changes in its parenchyma. Shown by Tiffany(11)and Lambert(13).

TABLE III

SIX CASES—CLINICALLY DIAGNOSED AS CEREBRAL ARTERIOSCLEROSIS. AVERAGE AGE 67.8 years

Autopsy No.	*Name*	*Age*	*Clinical Diagnosis*	*Pathological Findings*
1660	J. M.	60	Cerebral arteriosclerosis (confusion, poor memory, poor orientation, and fabrication)	Brain weight, 1,130 gms. Moderate cerebral atrophy; many senile plaques; marked neurofibrillar alteration; cerebral arteriosclerosis; thrombosis of left inferior frontal artery; chronic valvular endocarditis.
1670	M. F.	75	Cerebral arteriosclerosis, with attacks of confusion, vertigo and convulsive seizures.	Brain weight, 1,190 gms. Cerebral arteriosclerosis; slight cerebral atrophy; many large senile plaques; marked neurofibrillar changes; diffuse nephritis.
1680	T. P.	67	Psychosis with cerebral arteriosclerosis.	Brain weight, 1,275 gms. Cerebral arteriosclerosis; slight cerebral atrophy; chronic vascular nephritis.
1676	J. N.	77	Psychosis with cerebral arteriosclerosis	Brain weight, 1,070 gms. Cerebral arteriosclerosis; moderate cerebral atrophy; general visceral arteriosclerosis; diffuse nephritis.
1696	P. J. B.	60	Cerebral arteriosclerosis Psychosis	Brain weight, 1,400 gms. Moderate cerebral arteriosclerosis; slight cerebral atrophy; slight fibrillar thickening; no plaques; no nerve cell changes.
1705	J. D.	68	Cerebral arteriosclerosis	Brain weight, 1,325 gms. Subpial hemorrhage; cerebral arteriosclerosis; vascular nephritis.

Two cases (1660 and 1670) showed numerous plaques and marked neurofibrillar changes. These cases, even clinically, were regarded as impure cases of cerebral arteriosclerosis, and a few of the symptoms were explained on possible co-existing cerebral senile changes. One case (1696) showed thickening of the fibers but no plaques nor cell changes. The other three cases showed neither plaques nor neurofibrillar changes.

TABLE IV

Autopsy No.	*Name*	*Age*	*Clinical Diagnosis*	*Pathological Findings*
1661	E. K.	40	Unclassified	Brain weight, 780 gms. Marked cerebral atrophy; small senile plaques; marked neurofibrillar changes, whorled and snarled forms (Alzheimer's); chronic pulmonary tuberculosis; mili-tuberculosis of liver and spleen; chronic nephritis.
1715	H. G.	53	Manic depressive (depressed) phase Osteomyelitis	Brain weight, 1,475 gms. Edema; cerebral atrophy; many senile plaques; neurofibrillar changes; diffuse nephritis; osteomyelitis.

As to the nature of the plaques, they are best studied in the various silver preparations, preferably the Bielschowsky stain. A ripened plaque is composed of a central, dark, homogeneous nuclear-like mass from which there is a spoke-like radiation of similar material which eventually terminates in an outer condensed mass, see Fig. 1. Its constituents are explained in various ways by different observers, but apparently it is composed of degenerated nerve cells, nerve fibers, neuroglia tissue, and general cell débris.

These plaques vary in size, from that of a red blood cell to one of forty or fifty microns in diameter. They abound mostly in the second and third layer of the cortex, see Figs.

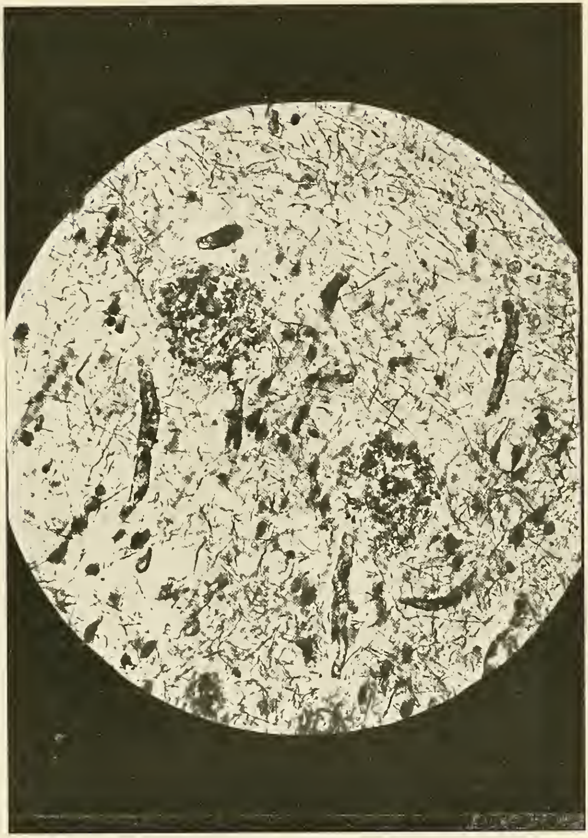

Fig. 1. Senile Psychosis

2 and 3, the ectal, polymorphous, and small pyramidal layers, though no layer is free from them. They are usually surrounded by a layer of neuroglia tissue, an attempt having been made by nature to wall them off as foreign bodies. The Herxheimer and Scharlach red fat stains show not only the presence of fat within these plaques, but also in the structure immediately surrounding them. The neurofibrillar changes are best described by Alzheimer (5), as follows: "In the interior of an otherwise apparently normal cell one or more fibrils, on account of increased thickness or impregnability, stand out prominently. Following this initial change many of the fibrils of the same cell running side by side are changed in the same way and are welded together to form a thicker and more darkly stained bundle which gradually comes to the surface of the cell. At last, the nucleus and interfibrillary protoplasmic substance of the cell disappears completely and only a bundle of fibrils wound into a snarl remains where before was a nerve cell," see Fig. 4.

As pointed out, these plaques occur most abundantly in the second and third layer of the cortex, the function of which is essentially that of association. The clinical symptoms of

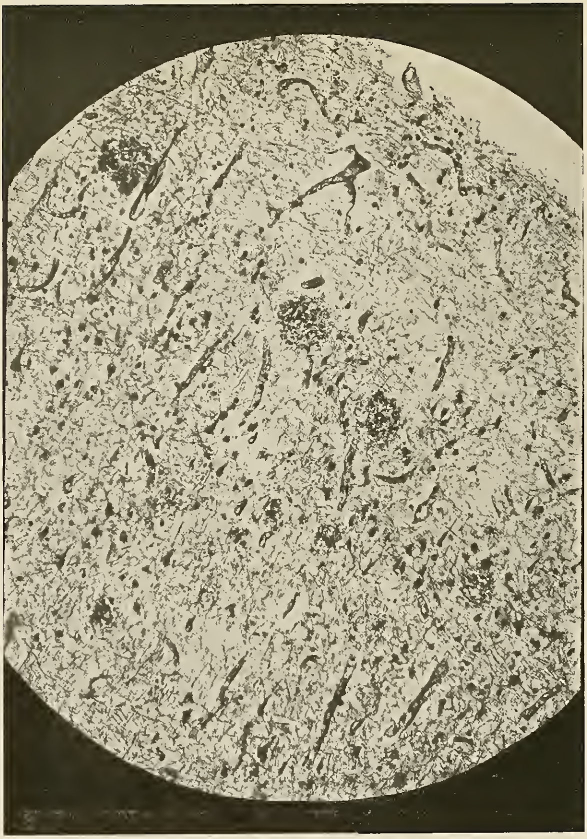

Fig. 2. Senile Psychosis

these psychotic individuals, lack of perception, poor memory for recent events, tendency to reminiscence, and general mental confusion —all could be explained by the failure of proper function of these cells of association.

Alzheimer (5), in 1906, described a condition occurring in a man in the fourth decade of life who presented a picture of an insidiously developing but rapidly progressive course resulting in profound dementia, with focal-like symptoms of aphasia, in whose brain at autopsy were found numerous plaques and the distinct neurofibrillar changes described above. In other words, these presenile psychoses occur in individuals whose nervous systems have terminated their life cycles rather prematurely, giving a clinical picture of inattention, indifference, and absent-mindedness in a man previously a fairly efficient worker. There is progressive impairment of memory, retention, grasp of current events, and poverty of thought, and an aimless restlessness with foolish behavior; eventually there is complete mental dilapidation and incapacity to comprehend, to talk, to walk and a sinking to a general vegetative existence. The case that follows typifies this.

The patient, E. K., was sent to the Man-

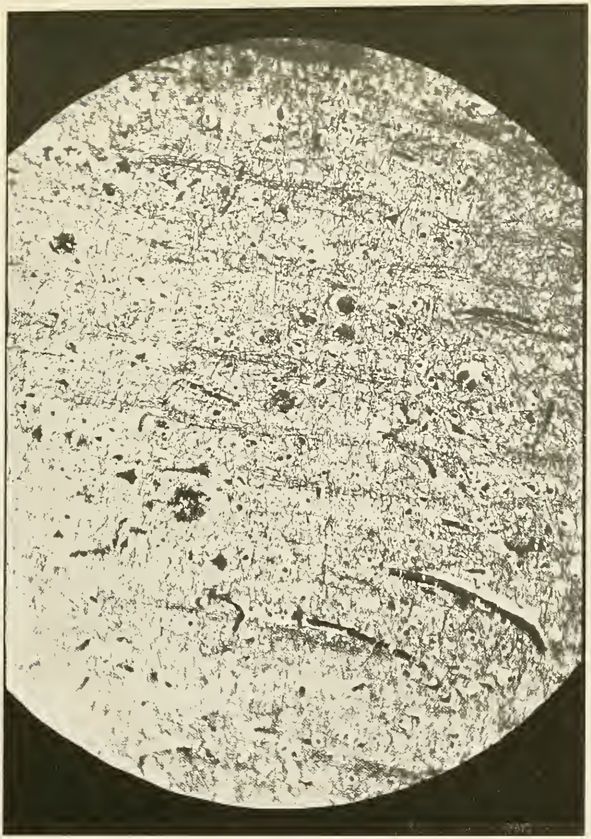

FIG. 3. SENILE PSYCHOSIS

hattan State Hospital September 26, 1917, and died on the 8th of the following December. No anamnesis could be obtained, owing to his condition; he was inaccessible, dull, demented, and confused. The only information obtainable, concerning him, came from the Department of Charities which merely gave his age as forty years and the statement that he had an alcoholic history. He had been sent to Bellevue Hospital for observation because he was violent and destructive; while there, he attempted to choke other patients, was resistive, restless, confused, and obstinate; talked in an irrelevant, rambling manner; was disoriented; delusional; and was considered to be an imbecile. His pupils were sluggish, the left one larger than the right. Knee jerks were increased.

Physically he was in fair condition, but was unable to stand or walk without assistance because of contracture of the knees; there was loss of sphincteric control; and the pupils were contracted and showed very little reaction to light. He was unable to feed himself and whined and cried whenever he was noticed. His speech was unintelligible. Throughout his hospital residence he remained in this condition, showing progressive

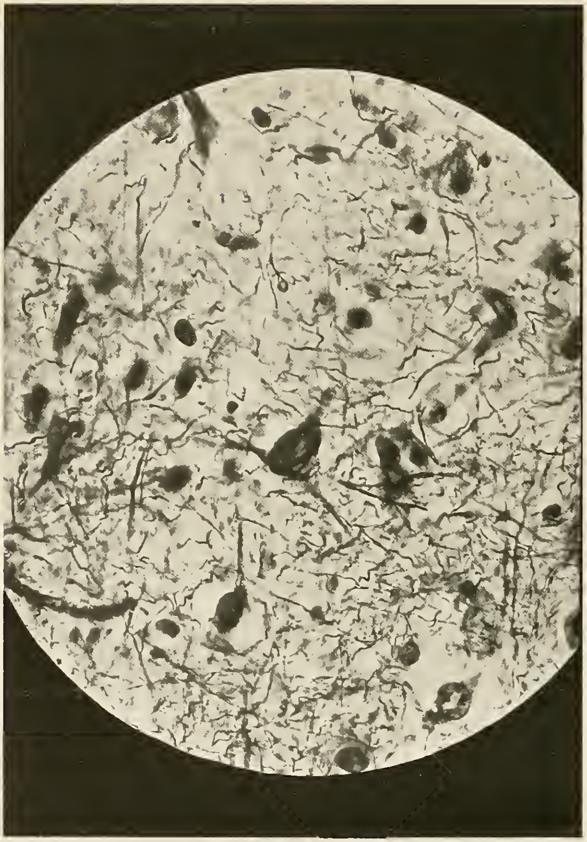

Fig. 4. Presenile Psychosis

physical failure, eventually dying of bronchopneumonia, three months after admission.

Autopsy was performed eighteen hours postmortem. The brain weighed 780 grams. It was generally contracted and very much smaller than normal. On incising the dura an unusually large amount of cerebrospinal fluid escaped. Over the temporal lobes there was definite atrophy, particularly near the tip where the fissures were very deep and wide and the convolutions quite narrow. The pia was generally thin and everywhere glistening. The vessels were of normal thickness. A horizontal section revealed a somewhat dilated ventricle and slightly atrophic cortex. Sections from the frontal, precentral, and gyri recti were examined microscopically. All showed a somewhat thickened pia but no cell increase. There was a definite loss of cells in the first three layers of the cortex with a corresponding increase in neuroglia. A section from the frontal and temporal convolutions showed small senile plaques. There were also numerous neurofibrillar alterations, whorls, and snarls.

The vessels of the spinal cord were somewhat thickened. Examination of the thorax revealed a right fibrinous pleurisy, apical tuberculosis, and left bronchopneumonia; the abdominal organs showed miliary tuberculosis of the liver and a chronic nephritis.

In this case, we had no satisfactory anamnesis and the onset of the illness is unknown. In the Manhattan State Hospital the patient showed extreme mental dilapidation, and was noisy and restless. Physically he showed complete loss of sphincteric control, sluggishly reacting pupils, and contracted hamstrings. He was placed in the unclassified group on account of unsatisfactory data and inaccessibility for mental examination. A postmortem examination revealing marked atrophy of the cerebrum with beginning plaque formation, and neurofibrillar changes, pictures conditions which would resemble those of a man who had suffered from senile psychosis, but which seen in a man in middle life could only be attributed to premature senility.

From these cases—twenty-eight of which were over sixty years of age, one forty, and one fifty-three; all having been psychotics, twelve of which were clinically diagnosed as senile psychotics, ten as dementia precoxes, six as arteriosclerotics, one as a manic depressive (depressed), and one unclassified—one might be justified in deducing a causal relationship existing between neurofibrillar changes and senile plaques on the one hand and the senile psychoses on the other.

I am indebted to Dr. M. B. Heyman for permission to present and publish these cases, and to Professor Francis Carter Wood for the microphotographs.

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ON THE NATURE OF MUSCULAR DYSTROPHIES, WITH A REPORT OF CHANGES IN CARDIAC MUSCLE IN TWO CASES

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Since Bell in 1830, Partridge in 1847, and Meryon in 1853 reported typical cases of muscular dystrophy, and Duchenne's claim that Meryon's cases were identical with the *paralysie atrophique graisseuse de l'enfance* was proven an error, there have been various and divergent opinions expressed concerning the pathology and etiological factors in cases of muscular dystrophy.

In spite of the occasional publication of postmortem findings in the central nervous system, certainly no constant nerve changes are described, and in most of those studied, there is reason to doubt the value of the observation or the correctness of the diagnosis of the clinical picture. We believe the primary myopathies, certainly the dystrophies, of themselves, present no anatomical changes in the peripheral or central nervous system. The dystrophies are in every way distinct from the degenerative muscular diseases, the pathology of which lies in the anterior horn cell of the cord and the analogous cells of the medulla. However, are we to consider the pathology of the primary myopathies as solely expressed by changes within the muscle structure? Are we to look for the etiology of these muscle affections to influences operating solely upon muscle or upon nerve endings or end plates? In the light of later researches disclosing the inter-relationship between body chemistry, the endocrine glands, and the autonomic system, are we not to reconsider critically the accepted interpretation of the primary myopathies, with progressive muscular dystrophy as its classic type, as an abiotrophy, in which muscle tissue alone is the element of defective organization?

A review of the publications of studies on the finer pathological changes since Meryon's early description of his findings as a picture of fatty and granular disintegration of voluntary muscle fiber, to later observations and reports of muscle fiber hypertrophy, pseudo and true, met with various interpretations as to the nature of the process; even observations on the finer pathology have not been uniform. For example, nearly all writers on the subject speak of true hypertrophy as sometimes observed. This is erroneous and confusing when the process of muscle hypertrophy is to be considered as part of a more extensive process. One does not see true hypertrophy of muscle fiber in the dystrophies. True hypertrophy, that is, physiological enlargement of muscle fiber, means an increase in sarcoplasm and sarcostyles; hypertrophy as seen physiologically after prolonged stimulation does not show the splitting and loss of angular contour of the fibers with connective tissue proliferation and characteristic arrangement of nuclei in the cell as seen in dystrophy. True, the fibers in dystrophy are often seen apparently enlarged. The process is doubtless of the nature of a swelling, and a part only of the general pathological picture within the muscle. In cases of dystrophy we rarely, if ever, see fatty degeneration. What we do see are adipose deposits. In a case but recently autopsied at the Montefiore Hospital, some of the large muscles of the trunk, especially of the rectus abdominalis, the sternocleidomas-

From the Wards of the Montefiore Hospital

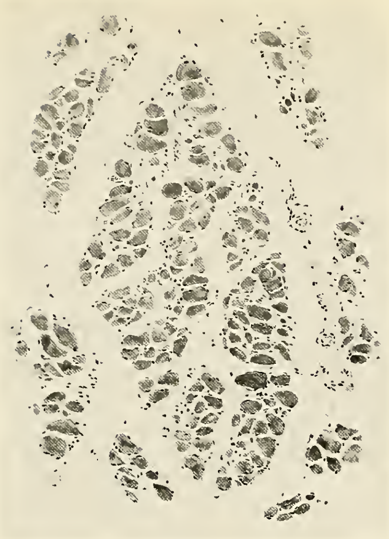


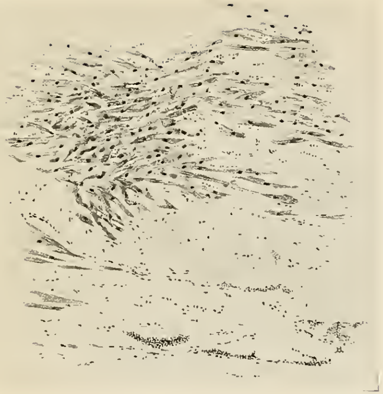
FIGURE A

CROSS SECTION OF SKELETAL MUSCLE, SHOWING MARKED INCREASE OF INTERSTITIAL CONNECTIVE TISSUE, AND A MODERATE NUMBER OF SMALL ROUND CELLS IN THE MESHES OF THE CONNECTIVE TISSUE

The muscle fibers show various degrees of degeneration. Some of them are markedly swollen, assuming a rounded form, while others are extremely small. The nuclei in either case are pyknotic.

toid, and the upper portion of the trapezius were really replaced by adipose deposits. For reasons which will presently appear we are slowly but surely coming to the conclusion that this almost anomalous behavior in pathology, this reaction of muscle tissue, is not an isolated one, but rather a far more generalized response to abnormal conditions within the organism. To consider the dystrophies and probably other forms of the primary myopathies as purely an abiotrophy is no longer justifiable.

In studying the pathology of skeletal muscle structure, cognizance has not been taken of the fact that striated muscles are not merely elements for contraction, serving the sole purpose of motility, but in addition are possessed of functions of equal importance, not unlike those of the organs of internal secretion. We know that muscle fibers in the process of their physiological activity elaborate a highly organized protein, with a pigment, strikingly similar to hemoglobin. Muscle tissue is constantly building up glycogen from glucose brought by the blood stream, and the glucose again is broken up into simple sugars, making these available for the muscle fibers' use in further physiological activity. It has been suggested (McCrudden) that the myasthenia of the myopathies and asthenia of other conditions in which we find hypoglycemia (Addison's disease, dyspituitarism, adrenalectomy, thyroidectomy, parathyroidectomy, phosphorus and hydrogen poisoning, and the toxin of diphtheria) are due to the reduced content of blood sugar. Janney, Isaacson, and McCrudden lay great stress upon disturbed metabolism, especially hypoglycemia, as associated with the primary myopathies. The primary influence may be looked for in dysfunction of the endocrine glands. The production of creatin and creatinin, and the elimination of sarcolactic acid and of carbon dioxide, all point to the conclusion that muscle is itself an important 'parenchymatous' organ, capable



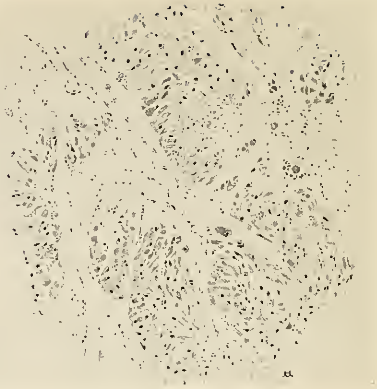


FIGURE B (Left)

SECTION OF HEART MUSCLE FROM MACROSCOPICALLY RECOGNIZED FIBROID SPOT

The muscle fibers here are seen fairly well preserved, but definitely shortened by encroaching connective tissue of scar formation. More complete description is given in the text.

FIGURE C (Right)

SECTION OF HEART MUSCLE SHOWING SCAR FORMATION AND CARDIAC MUSCLE FIBERS IN CROSS SECTION

Signs of degeneration of the muscle fibers are quite evident, especially in the fibers that have been isolated by the engulfing connective tissue. Most of the fibers are smaller than normal in size; their nuclei are pyknotic.

of function, in a way analogous to that of the true internally secreting glands. The similarity of activity is further suggested by the behavior of muscle in the elimination of metabolic products which are thrown into the blood stream to be carried to distant structures to inhibit or stimulate the latter. Thus, the muscle may be said to eliminate hormones. The probability is that the primary myopathies, including the dystrophies, Oppenheim's myotonia, and amyotonia atrophica have endocrine dysfunction as a direct or indirect etiological factor. A more careful study of these affections, especially the dystrophies, reveals them as associated with disturbances of the organism, involving metabolic processes and the osseous, trophic, and glandular systems, with manifestations identical with those observed in polyglandular affections. In the body chemistry in the dystrophies are found changes of the same nature as are seen in myxedema, Addison's disease, hypopituitarism, experimental thyroidectomy, etc. Among the metabolic manifestations, for example, hypoglycemia, now recognized as probably present in all cases of muscular dystrophy, is very frequently observed in myxedema, cretinism, exophthalmic goitre, Addison's disease, etc. The glucose of the blood is the source of contraction of muscle. The blood of the dystrophy patient can but poorly supply the muscle with carbohydrate, its necessary pabulum. Herein may lie the cause of muscle degeneration. McCrudden, and later, Janney, Isaacson, and the writers, have shown in their reports on nine cases the peculiar and constant behavior of carbohydrate metabolism in the dystrophies.

The fat found in the muscles of dystrophy cases is not primarily the result of degeneration of muscle tissue. Similar deposits of fat are found elsewhere in the body and again suggest lipomatosis of endocrine function. The roentgenologic picture of the osseous system is strikingly like that seen in endocrine disease, and the same is true of the hirsutes.

Creatin and creatinin are closely associated with muscle metabolism and the peculiarities of elimination of these we know are influenced by the endocrine organs, and in the dystrophies the behavior of these protein bodies again suggests the influence of the adrenals.

Herein we seem to have the explanation of the involvement of muscle structure in the myopathies. Practically no changes, at least no constant ones, are found in the nervous system in cases of primary myopathies.

It is rather surprising that affection of heart muscle in the myopathies has rarely been found. We are inclined to believe, however, that more careful search will disclose some changes of a character similar to those found in the skeletal muscles. The cardiac muscle, being histologically different and subserving different function, is doubtless different in its finer metabolism; hence, the changes are but slight. Of course, were it not so, the life history of the patient would indeed be brief. We are presenting two specimens of cardiac muscle (from two cases of muscular dystrophy), however, in which definite evidence of the involvement of the heart muscle is demonstrable. The skeletal muscle changes in these cases are those usually described in the autopsied cases. The microscopic appearance of the cardiac muscle in the two cases presented reveals some interesting features, and is the more important, since it suggests the same process as that seen in the skeletal muscle in muscular dystrophies. These would seem to challenge the usual statement that the heart muscle is almost never involved. True, a few observers claim to have found some changes, but their statements and descriptions are vague. It is improbable that we are dealing here with secondary changes. No Aschoff bodies were found in the numerous sections examined and hence the cicatricial tissue is probably not of infectious origin; neither in the ante mortem clinical findings nor in the history is there anything to account for the cardiac muscle changes. In the sections fibroid replacement of parenchyma is well defined; relatively large patches of fibrous tissue are found throughout the several microscopic fields. These scars are composed of dense white fibrous connective tissue; many small round cells and fixed connective tissue cells are found scattered throughout the cicatricial areas. Numerous heart muscle fibers are found isolated in small groups by strands of fibrous tissue. These muscle fibers appear to be engulfed by the scar tissue and much shortened by the encroaching granulations. While their cross striations are quite distinct, their nuclei show different degenerative changes approaching the stage of pyknosis. Then again, there are large islands of cardiac muscle fibers, which present at the periphery a peculiar fringelike appearance. The latter is caused by the infiltration of the connective tissue. Here the muscle fibers appear to be somewhat larger than normal in size, their angular contour being lost. While they have retained their cross striations, they nevertheless show degenerative changes, evidenced by pyknosis of the nuclei. Parts of the field, especially those in cross section, are strikingly like those seen in skeletal muscle in cases of muscle dystrophy, Figures A, B and C. Sections from the skeletal muscles showed the usual characteristic appearances found in cases of progressive muscular dystrophy and we need not reproduce them here.

The only demonstrable pathological findings involving the organs of internal secretion were observed in the thymus. This organ was found to be subinvoluted, as shown by its large size in both autopsied cases. (The patients were respectively 18 and 16 years old.) Microscopically, these organs presented an interesting feature as shown in the various stages of Hassall's corpuscle formation. In studying these corpuscles, one sees in the various stages a gradual transformation of a blood vessel into bodies made up of concentrically arranged endothelial cells. In the earlier stage there is but a slight thickening of the endothelial lining of the blood vessels with the contained blood coagulated and undergoing hyalinization. In the later transitional stages, the endothelium is seen to proliferate, giving rise to several layers of concentrically arranged cells, the lumen of the vessel in this way becoming narrowed until finally the entire structure is replaced by a well-formed Hassall's corpuscle.

The histories of these two cases are briefly as follows: H. B., a male; aged 18 years; father is an epileptic; two brothers of the patient are affected with muscular dystrophy; one maternal aunt is insane. The onset at the age of 5, was characterized by general muscular weakness and slowly progressive atrophy. Upon admission to the Montefiore Hospital in 1910 the calf muscles showed typical pseudo-hypertrophy, the orbicularis oris and palpebrarum muscles were atrophic, the former largely replaced by fat. Marked trunk deformities were present, due in large part to muscular atrophy. The hair was strikingly long and abnormally distributed; that of the scalp was peculiarly dry and brittle; there were extensive trophic ulcers on the fingers and toes. The roentgenographic examination showed a diffuse rarefaction of all the long bones; they were abnormally thin and under-developed. This peculiarity of the osseous system was observed in all nine of the series of cases studied at the Montefiore Hospital.

The second case, J. G., was a male, aged 16; family history in its bearing upon the case was negative. The essential features upon admission were most profound: general muscular weakness, atrophy, and contractures involving almost the entire musculature of limbs and trunk. The proximal muscles were especially involved. The distortion of the trunk was extreme. The case presents advanced muscular wasting with some evidences of pseudo-hypertrophy of calf muscles and deltoids. The facial muscles on both sides were involved, giving the typical myopathic facies. There was exophthalmos, marked dryness of the skin, and a deposition of brown pigment. The teeth were widely apart and there was also macroglossia. The roentgenographic examination revealed the same osseous peculiarities of retarded growth, etc., as above described.

REVIEWS & ABSTRACTS

NERVE WOUNDS: SYMPTOMATOLOGY OF PERIPHERAL NERVE LESIONS CAUSED BY WAR WOUNDS. By J. Tinel, M.D. 317 pages. 323 illustrations. William Wood & Company, New York, 1917. $4.50.

INTRODUCTION

The work of Professor Tinel, based as it is on a very intensive and extensive study of hundreds of cases of peripheral nerve injuries, is a distinct contribution to neurology. Its great value consists in the very lucid classification of the various types of injuries, which serves as a guide to correct diagnosis and appropriate treatment. Much of the material, of course, is not new, but what is new, and valuable, is the division of cases into typical syndromes, based on anatomy, physiology, pathology, and symptomatology. According to the author we can tell with certainty whether we are dealing with a case of complete interruption, compression, irritation, regeneration, or dissociation. Obviously, this is of vast importance in the matter of treatment, and the indications enumerated for guidance are valuable in the extreme. There are points, indeed, which might be justly criticized, as, for instance, the lax use of the term anesthesia for analgesia, etc., but as the object of this abstract is to present the excellent points of the book, criticism has been left out of the scheme.

Feeling that an accurate knowledge of nerve wounds is invaluable at this day, this abstract is published for the benefit of those who would rather study an epitomized form of the whole work. Although it is shortened, nothing has been left out which is necessary for a complete understanding of the subject. But the abstract must not be looked upon as a substitute for the main work; rather should it be used as a sort of *vade mecum* by those who have already studied the full text.

I. CAUSES OF NERVE WOUNDS

Direct and indirect: cutting, tearing, crushing, stretching, splitting, contusion, inclosure in cicatrix, callus, or hematoma. At the proximal end a *neuroma* may form, which consists of an entangled mass of axis-cylinders which meet with an obstacle to regeneration. A *pseudo-neuroma* forms at the distal end and consists of proliferated glia or laminated sheaths. The latter may not be an obstacle; the former always is and demands resection.

II. DEGENERATION AND REGENERATION

Wallerian Degeneration.—This occurs in the distal end. The axis-cylinder becomes fibrillary, splits into fragments and disappears. The myelin swells, becomes irregular, sinuous, beaded, splits into droplets and disappears. The cells of the sheath of Schwann proliferate and together with leucocytes absorb the myelin—an empty sheath remains. Slight retrograde degeneration may take place in the central end and even chromatolysis of Nissl's granules in the anterior horn cells.

Regeneration.—The axis-cylinders of the central end grow into the empty sheath of Schwann, and its directing influence is known as *neurotropism.* As the axis-cylinder progresses, it is covered by myelin. The rate of growth is one to two millimetres per day.

Defective Regeneration.—Where the ends are not apposed, the central end sends out axis-cylinders, but as they cannot pass they coil up and form a tumor-*neuroma.*

III. HISTOLOGICAL LESIONS CAUSED BY NERVE WOUNDS

Section.—The peripheral end has a true glioma of proliferated neuroglia and the cells of the sheath of Schwann are arranged in regular or irregular rows.

Complete Interruptions and no Break in Continuity.—Here we have, at the site of the injury, a fibrous cicatrix—nerve keloid of Dejerine—which bars the progress of the axis-cylinders. Centrally, there is the *neuroma;* peripherally, the *glioma.*

Pseudo-Neuroma Resulting from Bruising.—Here the axis-cylinder is not interrupted, but the myelin is broken and the axones may be fibrillated, beaded, or barbed; there is proliferation of glia and formation of a fibrous cicatrix. This gives the syndrome of compressions with temporary, incomplete, irregular, dissociated syndrome. The symptoms of irritation and trophic changes may be present. Regeneration is usually rapid.

IV. DISSOCIATED SYNDROMES AND PARTIAL LESIONS

The same nerve trunk may show part or all of the above lesions. There may be reflex sympathetic involvement—*irritation à distance.* Irritation and causalgia may result.

Diagnosis of Nerve Lesions

Clinical Examination.—Before intervention is resorted to, there should be a thorough muscular and cutaneous examination. There should also be an objective examination and a physiological exploration of the exposed nerve. The most efficient examination is accomplished by electrical exploration. Endeavor should be made to discover the presence of neuromata.

I. PRELIMINARY EXAMINATION AND HISTORY OF THE CASE

Examination of the wound should determine the entrance and exit of the projectile, the position of patient when the wound is received, the bone relations, and the existence of suppuration. The date of the wound and the time elapsing since it has occurred are valuable diagnostic points, as well as the history of the case. The most advantageous time for operation is two months after the injury. Paralysis is more extensive at the time of injury, atrophy appears late, pain from irritation comes on only after eight or ten days and increases up to a month, formication from pressure occurs only after four or six weeks. Reaction of degeneration appears after three weeks. Delay of two months is not against recovery.

Investigation of the first sequelae of the injury should determine whether paralysis was immediate or secondary. In the first case, it represents the cutting of the nerve; the second condition may be due to hematoma, cicatrix, callus, splints, or casts. Sudden sharp pains, numbness, cramps, contractions, each has its significance. The evolution of symptoms may be gauged by the increase or decrease of sensory or motor functions, trophic disturbances, presence of pain, numbness, and formication.

II. CLINICAL EXAMINATION

This examination should include a study of the attitudes in action and in repose. One must not confuse paralytic attitudes with functional inertia, psychic paralysis, contractures, or cicatrical contractions. Examination of voluntary muscles should differentiate between weakness and total loss of function. The part to be tested should be placed in position so that it will not have to work against gravity. In the case of psychic paralysis or functional inertia, response may be elicited by faradic current (except in some cases of compression). Care must be taken not to attribute the compensatory movements of neighboring muscles to the paralyzed muscles.

Examination of Reflexes.—In muscular paralysis the deep reflexes are abolished; in anesthesia, the superficial reflexes are lost.

Objective Examination.—Muscular atrophy comes on only after several weeks. It occurs very quickly after section, even more rapidly after irritation, but more slowly after compression. The atrophy may be due to immobilization or may result from bone, tendon, or joint lesion.

(*a*) *Muscle Tone* is lost in complete section, less in compression, and very slightly in irritation.

(*b*) *Mechanical Contractility*, the idio-muscular reflex, is present and even increased in paralysis. The reaction is slow but persists as long as galvanic irritability is present. Pain on pressure of the bellies of the muscle is absent in sectioned nerve and present in irritation. Fibrous contraction of muscles in nerve irritation leads to definite attitudes and postures and limited motion.

Trophic and Vaso-Motor Disturbances.—These are slight or absent in interruption and simple compression, and are almost constant in irritation. Glossy skin, disappearance of folds, dryness, dull color, and fibrous consistence of the integument are characteristic. In neuritis, there is excessive sweating.

(*a*) *Vaso-motor* disturbances manifest themselves in dryness and thickening of the skin and cyanosis, aggravated by cold and if the limb is dependent. Pressure of the finger produces a white spot on the paralyzed limb. Redness of the skin reaches an extreme degree in certain irritations.

(*b*) *Edema* and chilblains are also found in nerve interruptions. All lesions correspond exactly to the nerve distribution.

(*c*) *Ulceration* is rare in peripheral nerve lesions. These lesions are scarcely ever spontaneous; they are always due to pressure.

(*d*) *Thermal Disturbances.* In slight irritations, rise of local temperature is found. Cooling of a limb results from vascular lesions and is accompanied by cyanosis, edema, and progressive fibrous infiltration which characterizes ischemic paralysis.

(*e*) *Skin Appendages.* Hypertrichosis is frequent. In simple compressions, nails may be grooved transversely. In irritations, they may be striated, split, laminated, thinned at the edges, curved, atrophied, or conical. The fasciae may be thickened, contracted, or nodular; so also the synoviae. The joint may undergo fibrous ankyloses. Osseous decalcification is common in certain nerve irritations. Atrophy of the whole limb *en masse* may also take place. All these changes are common in irritation but are also found in ischemic paralysis—hence the necessity of differential diagnosis.

Objective Sensibility.—Study of the sensibility to pain, touch, and temperature, as well as deep sensibilities, should be made repeatedly and recorded on charts.

Objective Examination of the Nerve.—(*a*) *Pain on Pressure* is absent in complete section; it is present below the lesion in neuritis and neuralgia.

(*b*) *Formication on Pressure* is felt at the point of pressure, but if it is due to regeneration, it is felt in cutaneous distribution and is progressive. If new axis-cylinders strike an obstacle, there is fixity of formication. Formication sets in about four weeks after injury and persists during regeneration.

(*c*) *The Presence of a Neuroma* is always at the seat of a lesion, but it is not of great diagnostic value, even if found.

III. ELECTRICAL EXAMINATION

Faradic.—If stimulation of the nerve above the lesion does not provoke contractions in the muscles, then there is interruption. Simple hypo-excitability is judged by comparison with sound side. Inexcitability always accompanies complete peripheral paralysis. Return of faradic excitability comes after the return of voluntary movements.

Galvanic. The strength of current at the minimum closing contraction is the threshold of excitation. Normally, KCC>ACC. Sometimes, normally, the supinator longus, tibialis anticus and peroneals show polar inversion, ACC>KCC.

Syndromes of Nerve Interruption.—Reaction Degeneration (RD). Faradic and galvanic inexcitability of the nerve and faradic inexcitability of the muscle, as well as galvanic hypo-excitability at the motor point, with polar inversion. There is also muscular hyperexcitability (longitudinal reaction).

Syndromes of Compression or Irritation.—Partial RD.

Syndromes of Fibrous Transformation.—Muscular and galvanic hypo-excitability.

Reaction of Exhaustion.—This is comparable to Jolly's myasthenic reaction.

Myotonic Reaction. — Excessive galvanotonus is seen in recent RD. Disturbances of electrical reactions are seen in anterior horn lesions, root lesions, and nerve lesions, but never in functional paralyses or paralyses of cerebral origin. Grave cord lesions (Marie and Foix) may give electrical disturbances because of reaction on anterior horn cells below the lesion. Ischemic paralysis gives electric disturbances but the muscles have fibrous transformation and they are more inexcitable than the nerve.

IV. ELECTROPHYSIOLOGY

(1) The active pole is always the negative pole at the closing of the current.

(2) The muscle deprived of its nerve is galvanically hyper-excitable.

(3) The velocity of excitability or *chronaxie* as means of electro-diagnosis.

Polar Action.—The negative pole alone causes closing contraction. The positive pole, on the surface, causes a virtual negative in the tissues, which is *diffuse* and acts *à distance*. Normally, to stimulate the motor point, say, one to two milli-amperes are needed by the negative pole, and five to eight milli-amperes are needed to stimulate the muscle itself (longitudinal contraction). The positive pole needs eight to ten times as much current as the negative for the motor point, but no more than the negative for the muscular belly because of the virtual negative produced deep in the tissues. Paralyzed muscles have no motor points; hence, the belly is more excitable. Muscle fibers are best excitable near the tendon (longitudinal reaction). This is not the descent of the motor point, as it is usually designated. The positive electrode (virtual negative) is equally efficient. Above the motor point, the negative is weak, while the positive is strong (virtual negative); hence, we get what is called polar inversion.

Hyper-excitability of the Paralyzed Muscles. Galvano-tonus.—The paralyzed muscle needs less current with the negative pole (longitudinal reaction). For the positive pole less current is needed than in healthy muscle.

Velocity of Excitability. Chronaxie.—The threshold of contraction—minimum intensity and minimum duration. The increased duration does not matter at all, if there is no change in intensity. Lessen the minimum duration of liminal intensity and there is no contraction, unless the intensity is increased. The velocity of current is the minimum duration of liminal current at the threshold. Chronaxie is rapid in the normal muscle, the intensity being 1/1000 second; in the paralyzed muscles, it is 40/1000 or 50/1000 second.

IV. CLINICAL TYPES

Syndrome of Interruption.—Comple section, severe compression, fibrous cicatrix. Distal end degenerates (Wallerian).

(*a*) Immediate, complete, invariable paralysis of all muscles—fixity of paralysis.

(*b*) Rapid, progressive disappearance of tone, before onset of atrophy.

(*c*) Progressive muscular atrophy. Absent tendon reflex; present idio-muscular.

(*d*) Gradual RD and complete in two or three weeks. Muscles lose faradic excitability; motor point disappears; polar inversion; slow galvanic contraction.

(*e*) Immediate, complete, invariable anesthesia. Deep sensibility is less extensively involved.

(*f*) Absence of spontaneous or induced pains and insensitivity of nerves and muscles.

(*g*) Absence of formication by pressure on nerves below lesion; presence at level of lesion (neuroma) and its fixity there. This comes on four weeks after injury.

(*h*) Absence of trophic change, except little edema, cyanosis, or hypertrichosis; occasionally trophic ulcers following injury.

Syndrome of Compression.—This is caused by a lesion which prevents passage of voluntary or electric impulses, but does not cause peripheral degeneration of axis-cylinder. May be temporary or partial compression and some interruption.

(*a*) More or less complete paralysis—almost as in interruption.

(*b*) Muscular atrophy—more rapid and less intense than in interruption.

(*c*) Relative preservation of muscle tone. Marked idio-muscular reflex.

(*d*) Partial, incomplete RD; slow in coming ou. Paradoxical reaction, *i. e.* Faradic responds below lesion and none above.

(*e*) Variable and less intense anesthesia and no fixity.

(*f*) Absence of formication; where present and fixed at level of lesion, shows insuperable obstacle and probable peripheral degeneration.

(*g*) Absence of trophic disturbance.

Syndromes of Irritation.—May or may not be paralysis.

(*a*) Serious nerve irritation occurs in mixed nerves; always some paralysis; not complete; partial RD. Atrophy variable. Tone is preserved; idio-muscular reflex present; fibrous infiltration of muscles. Pain and trophic disturbances occur later, progress for a few weeks and remain stationary for months; slowly disappear. The pains are spontaneous, burning, pricking, tearing and are intensified by emotion, heat, cold, friction. Pain on pressure of nerves and muscles. Cutaneous hyperalgesia, particularly to pressure, even in case of tactile and thermal hypoesthesia.

Trophic disturbances: edema, cyanosis, hypertrichosis, dry skin, scaly, fibrous or sweaty, or glossy. Nails are curved, furrowed, split, cracked, claw-like; conical atrophy of digital extremities. Fibrous infiltration of muscles, synovia, contraction of tendons, *griffes*, ankyloses, deformed joints, decalcification. Paralysis and pains gradually disappear but contractures are refractory. With disappearance of pain formication sets in and betrays advancing regeneration.

(*b*) Attenuated neuritic type. Slight pain, fibrous infiltration, contractions, cutaneous changes, or no cutaneous changes and contractions; *e. g.*, pes equinus in sciatic lesions. Sensory changes may be absent.

(*c*) Neuralgic type. Following slight bruises of nerve trunks. No trophic disturbances except slight skin changes; no paralysis but little weakness and atrophy, no electric changes, hyperesthesia, radiating pains on extension of limb. Pain on pressure of nerve trunk. They are persistent and disappear spontaneously.

(*d*) Intense neuralgia. Causalgia of S. Weir Mitchell. May affect all nerves but mainly median and sciatic. Usually violent burning located at end of nerve; *e. g.*, palm or sole. Movement aggravates, and so does all cutaneous excitation and even emotions; wet relieves. There are slight motor or vasomotor disturbances, a warm red, glossy skin and slight nail changes. Extreme hyperesthesia exists over a surface greater than area of nerve distribution. Paroxysms, emotional pains, vasomotor and sensory changes beyond area of nerve distribution speak for a sympathetic syndrome.

Syndromes of Regeneration.—There are (1) Signs of nerve regeneration: (*a*) Appearance of formication called forth by pressure. (*b*) Return of electrical conductivity. Former is early; latter very late. Progress of formication shows growth of axis-cylinders and begins fourth to sixth week. Absence of formication—absence of regeneration.

(2) Signs of muscular regeneration. Appearance of muscular tone; return of muscular sensibility and diminution of atrophy.

(3) Modification of electric and voluntary movement. Electrical reactions: Galvanic excitability increases; negative pole becomes positive; slowness of contraction diminishes. Faradic excitability returns last, after voluntary movement, with thick coil; before voluntary movement with thin coil.

(4) Sensory signs of regeneration. (*a*) Spontaneous formication. (*b*) Narrowing and disappearance of anesthetic zones. Deep sensation appears first; gross before finer sensations. (*c*) Paresthesia. Intense and disagreeable formication produced by skin excitation. They are diffuse; persist after the stimulus and precede return of sensation.

(5) Disappearance of trophic disturbances such as fibrosis, contraction, joint affection, etc., is late and often not at all.

Dissociated Syndromes.—Fascicular destruction may give in the same nerve partly signs of interruption, partly signs of regeneration, or different motor and sensory phenomena.

Ascending Neuritis.—This is a complication, often due to a pus focus and ascent of toxin in nerve or sheath. Central end degenerates. Extremely painful; violent; paroxysmal, painful on pressure all over course of nerve. The spread upward may reach the plexus, affect the cord, and even go to the opposite side. There is progressive ascent of atrophy, paralysis and anesthesia. The condition is very serious, obstinate and progressive despite treatment.

PARALYSIS, HYPERTONIA, AND CONTRACTIONS FROM NEURITIS

Slight irritative lesions often lead to marked and permanent contractions. Although all have a functional element, they are to be distinguished from purely functional cases by the trophic changes in skin and nails, sweat, slight sensory changes, and electric modification. Dread of pain, inaction and inertia help to form the contraction. The contractions are paradoxical; *i. e.*, the nerve lesion gives the opposite of the paralytic syndrome. There may be reducible or irreducible contractions, usually painful on forcible reduction and even if reduction is successful, there is a return of contraction in a short time.

Symptoms—(1) Slow in coming on. (2) Slight atrophy. (3) No electrical changes, or at most, slow contractions. (4) Mechanical hyper-excitability of muscles and exaggeration of tendon reflexes. (5) Trophic and vasomotor changes: Cooling of limb, profuse sweats and red skin, corresponding to the nerve region, or dry and pale skin, fibrous infiltration and hypertrichosis. The nails may be dull, yellow, striated and the pulp hypertrophied. (6) Almost always pronounced sensory disturbances — mostly hypoesthesia; rarely hyperesthesia. Often pain and formication on pressure on the nerve. (7) The contractions and other changes always have an anatomical topography. Slight injuries to roots of plexuses may give these neuritic symptoms, so also injuries to fingers or toes may give ascending neuritis and lead to contractions of muscles supplied by the nerve involved. Frost-bite may result in neuritic skin and nerve and muscle symptoms. There always is a functional element in these cases and immobilization and inactivity insure contractions.

Treatment—Gentle massage, general mobilization, hot faradic bath, perseverance and good will on the part of the patient.

General Diagnosis of Peripheral Nerve Lesions

Central Paralyses. — Spasticity, no atrophy, no electric disturbances. (*a*) Cord lesion; compression; crushing; hematomyelia. Same spastic symptoms. If roots are included in the compression then there are peripheral signs. A hematomyelia may be limited to the grey matter of cord; *e. g.*, first and second sacral segments may simulate sciatic paralysis. The sensory changes will be segmental and dissociated.

(*b*) Peripheral spontaneous polyneuritis may give the same symptoms as traumatic cases, but usually are symmetrical.

(*c*) Root inflammation due to meningeal irritation; always sensory or sensory-motor; seldom purely motor. Root distribution. Due to syphilis, tuberculosis, or cerebro-spinal meningitis. Pain on coughing and sneezing. Lumbar puncture shows signs of inflammation.

Functional Paralyses.—(1) They are paradoxical and out of proportion to the cause. (2) They are extensive, absolute. (3) Are not anatomical and paralyze a function, not a movement. (4) Paradoxical anesthesia, not anatomical but defined areas, segments or limbs. (5) No muscular atrophy, loss of tone, disturbed reflexes, or trophic changes. (6) Slight or no electrical changes. (7) Special attitude of mind; no effort; no interest. Treatment: Isolation, psychotherapy, faradism, re-education. Functional paralyses may result from disuse, fear of pain, pseudoparalysis from contraction of the antagonists, prolongation of organic paralysis after regeneration, or paralysis (auto-suggestive) due to anesthesia of a limb or part of it; *e. g.*, median.

Other Paralyses.—(1) From pseudo-arthrosis. (2) Resulting from muscular destruction. (3) Pseudo-griffes due to muscle or tendon contraction. (4) Due to cicatricial adhesions of tendons. (5) Reflex muscular atrophies due to fracture. (6) Due to hypotonia and muscle stretching; *e. g.*, pseudo musculo-spiral, prolonged postures.

Ischemic Paralysis.—Due to vascular obliteration; *e. g.*, ligature or thrombosis of great arteries; subclavian, axillary, brachial, femoral, or popliteal; also after tight bandaging. Reaction depends on unknown factors. It is hard to distinguish from *syndrome of irritation of nerve lesions.* There are two phases: (1) Edematous infiltration, cyanosis, and cooling. (2) Two or three months later, fibrous transformation of this edema. Progressively, the skin, muscles, tendons, joints are infiltrated with sclerous tissue, and the limb is hardened to a bat; the skin is glossy, desquamating, cold, purple, or red; nails deformed, bones decalcified, limb atrophied, fingers fusiform, bone atrophied. The pulse disappears from the part, the blood pressure is diminished or absent in the limb. Ischemic paralysis is almost always painful—burning, formication, tingling; deep-seated sensibility and pain on contact with cold or heat. There is superficial loss or diminution of sensibility. There are changes in electrical reaction even up to RD. Ischemic paralysis is often accompanied by nerve lesion and thereby aggravated. Differentiation from nerve lesions is often difficult.

Diagnosis—(1) No peripheral typography. (2) Segmentary or centrifugal distribution and worse at distal end of limb. (3) They are more severe, refractory, and often incurable. Hope lies in hot baths, massage, hot wrappings, mobilization, galvano-faradic baths and prolonged (months to years) patient treatment.

PART II

MUSCULO-SPIRAL NERVE

Arises from posterior cord of the brachial plexus. Paralysis of musculo-spiral nerve is most common. Callus from fracture; 'crutch palsy'; 'Saturday night palsy'; pressure of sharp edge against posterior surface of arm.

Supplies.—In axilla, triceps. (2) In bicipital furrow, supinator longus, and extensor carpi radialis longior. (3) Near neck of radius, extensor carpi radialis brevior and supinator brevis. (4) Posterior surface of arm, extensor carpi ulnaris, extensor communis digitorum, extensor proprius digiti minimi, extensor ossis metacarpi pollicis, extensor longus and brevis pollicis, and extensor indicis, also a twig to abductor pollicis (median thenar).

Sensory.—An internal cutaneous branch to axilla and postero-internal part of arm, an external cutaneous branch to posterior surface of arm and forearm, an anterior branch to external surface of thenar eminence, dorsal surface of thumb, and of hand up to second phalanx of index and half middle finger. (Sensory unimportant.)

Motor syndrome.—Paralysis of extension of forearm, wrist, hand, first phalanges, and thumb. Adduction is weakened (extensor carpi ulnaris). Paralysis of supination (supinator brevis). Paralysis of supinator longus, weakens flexion of forearm and the muscle is not felt in flexion. This is characteristic of peripheral lesion and not of root. The sign is absent in lead palsy, spinal cord lesion, polyneuritis, and hysteria.

Sensory Syndrome.—Restricted area of hypesthesia in dorsal surface of thumb, part of index finger and adjacent area of hand.

Trophic Syndrome.—Dorsal swelling of carpus frequently seen, due to relaxation of tendons, subluxation of os magnum, semilunar, and to a tenosynovitis. In neuritic forms the skin and wrist and finger joints show involvement. The posterior branch of the musculo-spiral has the trophic rôle. Nail and vascular changes are rare. Edema of the dorsum is rarely seen.

Total Paralysis.—Shows all symptoms. Olecranan reflex is absent. Lesion high in axilla.

Partial Paralysis.—Triceps intact and reflex present. Lesion high up. Above supinator longus: Same as above. Usually due to fracture, callus, or direct injury to nerve. Syndrome of irritation particularly common.

(*a*) Below supinator longus: Fracture of epicondyle or lesion at bicipital groove. The supinator and extensor carpi radialis longior are intact. Not to be mistaken for functional paralysis. The electrical reactions decide.

(*b*) Below radial extensors: Lesions at neck of radius or below. Extensor carpi ulnaris is still paralyzed; hand deviates to radial side on extension. Finger extension paralyzed. Dissociation of paralysis of extensor communis by a low lesion may leave extensor of middle finger intact. Fascicular topography of musculo-spiral shows outer fibers supply supinator longus and radial extensors, inner fibers, the extensor carpi ulnaris, muscles of thumb, extensor communis and supinator brevis.

Syndromes of compression.—Muscle tone partially preserved, wrist drop slight, atrophy slight, RD. incomplete. Recovery fairly quick.

Syndrome of Interruption.—Hypotonia and drop complete; rapid RD.; complete muscular analgesia. Pressure on the nerve at the level of the lesion gives formication at thumb.

Syndrome of Irritation.—Frequent, unaccompanied by pain, but prognosis is bad on account of tendon adhesion and joint limitations. Sensory disturbances are few. The drop is incomplete, fingers are extended, due to fibrous adhesions of the tendons, and flexion is impossible. Skin infiltrated, shows fine desquamation, swelling of articulations.

Syndrome of Regeneration.—First supinator longus, then radial extensor, and, finally, other extensors, thumb being last to recover. Complete supination is latest.

Diagnosis—(1) Destruction of the muscles of the forearm. (2). Hypotonia and lengthening of the muscles of the extensors due to injuries of the antibrachial muscles, resulting in prolonged flexion. Faradization and will movements are possible. (3) Hysterical paralysis of the hand usually simulates musculo-spiral, but flexion also is impossible, and there is segmentary anesthesia, no RD., etc.

Treatment—General. Splints and devices to keep up extension are very important.

ULNAR

Eighth cervical and first dorsal. Inner cord of brachial plexus.

Supplies.—Motor. (1) Twig to flexor carpi ulnaris. (2) Two branches to internal fasciculi of flexor profundus digitorium. (3) The three muscles of hypothenar eminence. (4) All interossei, palmar and dorsal and two inner lumbricales. (5) Adductors of thumb. (6) Inner head or flexor brevis pollicis.

Sensory.—Palmar: Little and half ring fingers, inner side of wrist, and hypothenar eminence. Dorsal: Inner surface of wrist and hand; little, ring and part of middle finger, last phalanx being supplied by the palmar branch. Vasomotor, trophic, and articular to elbow and carpus, ulnar artery, palmar aponeurosis and interosseous spaces.

Motor Syndrome.—Flexion of wrist and adduction of hand are weakened (Fl. carp. ulnaris.) Diminished flexion of fourth and fifth fingers (two heads of flexor profundus). Atrophy and paralysis of the muscles of the hypothenar eminence. Paralysis of interossei: Loss of flexion of first phalanx and extension of last two, except a little the two fingers served by lumbricales (median). Loss of abduction of fingers (palmar interrossei) and abduction (dorsal interossei). The flexors and extensors of fingers serve a little as adductors and abductors. If the hand is held flat on table, no abduction or adduction is possible. *Little finger cannot be adducted at all.* Prehension sign of Froment: adductor of thumb (ulnar) opponens (median) shown in seizing a piece of paper.

Sensory syndrome.—Palmar: vertical line through ring finger up to and above wrist. Dorsal: vertical line through middle finger up to and above wrist.

Trophic syndrome. — Atrophy, occasional dislocations, whole palmar surface may be affected. Desquamation and ulceration. Redness, cyanosis, neuritic fibrosis.

I. SIMPLE COMPRESSION AND RECENT INTERRUPTION

There is a great amount of substitution for the ulnar. Flexion of fingers is possible with the superficial and two external tendons of deep flexors, a little separation of fingers by means of extensors. Adduction is weak. Adduction of thumb very poor, but opposition is possible. Ulnar griffe is scarcely perceptible. Hand is flattened, hypothenar and adductors of thumb are flattened. Little finger flexed, also ring finger. The griffe becomes more marked after a time. In injuries below the epitrochlea (after branches to profundus are given off), the griffe is more marked; also in regeneration. The griffe is reducible.

II. PROLONGED COMPLETE INTERRUPTION

More common than compression. Characterized by rapid atrophy and hypotonia; fixity of sensory disturbance, early RD., fixity of formication; analgesia of nerve and muscles. Hyperextension of first phalanges; laxity of articular ligaments. Usually marked two-fingered griffe—possibly also middle and even index finger. The griffe is usually reducible. Trophic changes are absent. Vascular disturbances (cyanosis); dry skin; accidental ulcer may occur, limited to ulnar distribution. Marked atrophy of hypothenar, interossei, adductors of thumb, and flattening of thenar.

III. SYNDROME OF NERVE IRRITATION

Marked fibrous, irreducible griffe. Spontaneous pain, pain on pressing nerve trunk, antibrachial muscles, hypothenar eminence, and adductors of thumb. Painful anesthesia or hyperesthesia. Trophic disturbances; scaly skin; infiltration; split and rapidly growing nails; fibrous contraction of tendons, adhesions and contractions of palmar aponeurosis.

Paralysis may be slight. Slight irritation may give 'accoucheur' hand.

IV. NEURALGIA OF THE ULNAR

Not common. Slight irritation, no motor paralysis, very painful, but not so bad as median causalgia.

V. DISSOCIATED SYNDROMES

Fascicular topography. The flexor carpi ulnaris, profundus and adductors of thumb form the outer fibers and interossei, hypothenar muscles and sensory fibers form the inner fibers of the nerve.

Diagnosis of Ulnar Paralysis

1. Frequent absence of griffe, particularly in complete paralysis.
2. All ulnar movements, except adduction of little finger may be substituted.
3. Simple cicatricial contraction of flexor of little and ring finger may simulate griffe. The griffe is easily reduced.

I. CONTRACTIONS RESULTING FROM SLIGHT NEURITIS OF THE ULNAR

Accoucheur's hand.—Extension of fingers, adduction of little finger and thumb; passive moments are possible. No electrical changes; slight trophic and sensory disturbances. Voluntary flexion is possible; elastic resistance to passive flexion. Flexor contraction: Only of first phalanges if interossei alone are involved or of all phalanges if profundus and aponeurosis are affected. Most, if not all these contractions are the result of immobilization aided by indifference and the functional element. Therefore, always mobilize, massage, bathe, faradize, etc.

II. MEDIAN

Outer head together with musculo-cutaneous represents sixth and seventh cervical; inner head together with ulnar represents eighth cervical and first dorsal. It supplies: Pronator quadratus and pronator radii teres, flexor carpi radialis, superficial flexors, external two heads of profundus. Abductors of thumb, opponens, flexor longus and part of flexor brevis pollicis, external two lumbricales.

Sensory.—External part of palm to thumb and external half of ring finger. Dorsally, last two phalanges of index and middle and half of ring fingers.

Trophic.—Palm little affected, mostly index and middle finger; also nails; atrophy of thenar and lower part of forearm.

COMPLETE PARALYSIS OF MEDIAN ABOVE THE EPITROCHLEAR

Revealed only by movement; not at rest. Loss of pronation; loss of flexion of thumb, index and middle finger, weak flexion at wrist. Flexion of middle finger may be possible (ulnar). Only last two phalanges are affected.

Signs.—On making a fist, thumb and index finger remain extended; intertwining fingers, index is extended. Hand flat on tablet, cannot scratch it with nail of index finger. Sometimes, pronator radii teres, flexor carpi radialis and palmaris longus are not so strongly affected or regenerate much earlier, possibly because of anastomosis with musculocutaneous.

PARALYSIS OF MEDIAN IN THE FOREARM

Only thenar eminence is involved and sensory changes are present. Flexion of thumb is not affected. Opposition is lost but hard to bring out—thumb does not meet pulp of fingers, but their sides.

DISSOCIATED PARALYSIS OF MEDIAN

Lesions at external border affect pronator radii teres, flexor carpi radialis, flexor pollicis and opponens, but spares flexor of fingers. In lesion of internal part of nerve, only flexion of index and middle finger is affected, thumb and wrist being spared.

NEURITIS OF MEDIAN

Differs from causalgia in the marked trophic changes. Spontaneous pain, acute pain on pressure of nerves and muscles, painful hypo- or hyperesthesia. Trophic changes ending in median griffes. Nails of thumb, index, and middle fingers are striated, claw shaped and digital pulp is present. Griffes may result in flexion of thumb, distal phalanges of index, and middle finger, and irreducible contractures, or there may be irreducible extension of the distal phalanges (contrary to proximal phalanges in musculo-spiral).

CAUSALGIA OF THE MEDIAN

Very common. No paralysis or even anesthesia and few trophic signs. Comes on soon after wound and increases to maximum up to ten or twenty days. Terrible, persistent, paroxysmal *burning* pain. Cold, heat, touch, and dry air aggravate; wet relieves. Movement of limb and emotion aggravate or recrudesce. Insomnia, anorexia, emaciation, gloominess, solitude characterize the victims. They protect their hands. On examination: immobilization on account of pain, but no paralysis; light touch is painful; deep pressure is painless. Skin is smooth and glossy; no fibrosis. Nails are curved but not thick, split, or striated. After a few months, atrophy of the tips of fingers and tapering nails appear. Skin is red and moist. Sweats, vaso-dilator and constrictor signs and emotional pain point to sympathetic involvement, as do reflex vasomotor changes in cervical sympathetic. Condition lasts up to fifteen months. Treatment is unsatisfactory. Positive galvanic pole and radiotherapy have some effect. Nerve sections may be tried in very refractory cases.

Diagnosis.—Wounds of flexors in forearm may cause paresis or, if nerve is cut, simply anesthesia and functional paralysis. The flexors show no RD. and faradization convinces the patient that his power is there though the fingers are anesthetic.

ASSOCIATED PARALYSIS OF MEDIAN AND ULNAR

Caused by lesions up in the arm, atrophy of all flexors, thenar, and hypothenar and interossei. Flat hand. Hyperextension of wrist and first phalanges. Substitution flexion of wrist by extensor ossis metacarpi pollicis and extensor brevis pollicis. In regeneration, flexible four-fingered griffe occurs. Simultaneous irritation of ulnar and median gives fibrous griffe—ape-like hand, atrophy of thenar, hypothenar, and interossei.

MUSCULO-CUTANEOUS

Outer cord. Fifth and sixth cervical. Branches to coraco-brachialis, biceps, and brachialis anticus and sensory to antero-external and postero-external part of whole forearm, also anastomotic branch to median (motor).

Paralysis of all flexors of forearm except supinator longus. It may be overlooked unless biceps is tested. Overlapping reduces the sensory area to a narrow strip on the antero-external surface of the forearm and wrist.

CIRCUMFLEX

Posterior trunk. Fifth cervical. Supplies deltoid (twigs to subscapularis) and a sensory branch to tip of shoulder and external surface of arm. Paralysis is caused by direct trauma, fracture of neck of humerus and dislocation. In latter case it may be direct tear of roots of brachial plexus. External (acromial) fasciculi; anterior (clavicular) fasciculi; and posterior (scapular) fasciculi. Movement of shoulder becomes impossible; slight substitution only is possible with supra-spinatus. Flattening of shoulder. Small area of hypoesthesia on the external surface of shoulder.

INTERNAL AND LESSER INTERNAL CUTANEOUS

Usually affected together, often accompany lesions of ulnar and median in arm. Internal cutaneous supplies inner part of arm and forearm to wrist, anterior and posterior. Lesser internal cutaneous supplies posterior internal part of arm, except a triangular area above and behind which is supplied by second and third intercostal.

BRACHIAL PLEXUS

The roots lie between scalenus anticus and medius; the trunks in supra-clavicular fossa; the cords behind the clavicle and the nerves of the plexus in axilla.

Branches.—(1) To rhomboids from fifth cervical root. (2) To serratus magnus from fifth and sixth cervical roots. (3) Supra clavicular to supra and infra scapular from the upper trunk. (4) Upper branch to subscapularis from upper trunk. (5) Branch to subclavius from upper trunk. (6) Nerve to pectoralis major (external anterior thoracic) from upper cord. (7) Lower branches to subscapularis from posterior cord. (8) Nerve to teres major, from posterior cord. (9) Nerve to latissimus dorsi from posterior cord. (10) Nerve to pectoralis minor from lower cord (internal anterior thoracic).

LESIONS OF THE BRACHIAL PLEXUS

(1) Radicular syndromes from lesions of roots or primary trunks, in supraclavicular

area or near scaleni or vertebral column.

(2) Plexus syndromes from lesion of secondary cords in clavicular region or upper part of axilla.

(3) Radicular syndromes (roots and primary trunks) show motor and sensory root distribution. The paralyses are partial, as several roots supply one muscle. Anesthesia is also less extensive, as there often is substitution. Hence, three groups: I. Fifth and sixth cervical or upper trunk. II. Seventh cervical or middle trunk. III. Eighth cervical and first dorsal, lower trunk.

I. UPPER RADICULAR GROUP (ERB-DUCHENNE SYNDROME)

Shows paralysis of following muscles: (1) Deltoid (circumflex). Biceps and brachialis (musculocutaneous). Supinator longus (musculospiral). No flexion of forearm is possible. (2) Pectoralis major (clavicular head only). Supra and infraspinatus. Subscapularis. Teres major.

If the lesion is near the roots then the serratus, rhomboids, and levator anguli scapulae are affected. There results then atrophy of all scapular muscles, displaced scapula (winged) and no balancing of it to compensate for deltoid. (3) Partial coraco-brachialis, triceps, radial extensors and supinator brevis, pronator radii teres and flexor carpi radialis, extensors and flexors of muscles of thumb.

(4) *Sensory.*—Parallel to axis of limb: external, anterior, and posterior down to thumb, C–5 and C–6; supinator jerk over styloid is absent.

II. MIDDLE RADICULAR SYNDROME

Paralysis of musculospiral except supinator longus. The triceps is very weak. Feeble movements of extensors and extensor ossis metacarpi pollicis (partial C6) and extensor indicis and minimi digiti (C6 and C8). Looks like lead paralysis with similar integrity of supinator longus. The sensory region of the seventh cervical is restricted to a small tract over dorsal surface of forearm and external dorsal surface of hand. Olecranon reflex is absent.

III. LOWER RADICULAR GROUP (ARAN-DUCHENNE SYNDROME)

Eighth cervical and first dorsal give paralysis of flexores digitorum, flexor carpi ulnaris, interrossei, thenar, and hypothenar eminences. Eighth cervical goes to median, ulnar is principally first dorsal. Simian griffe of associated median and ulnar. Abductor pollicis gets fibers from C7 and C6, adductors of thumb mainly from C8, interossei mainly D1. Pronator radii teres mainly from external root of median, C6 and C7, hence, largely unaffected. Sensory root: band of hypesthesia on inner side of limb. Ulnar reflex absent. Klumpke paralysis: Oculo-pupillary symptoms in tear of D1 root.

Total radicular paralysis shows sensory and motor involvement of limb, oculo-pupillary signs, except normal sensory triangular area on inner side of arm, D1 and D2, and top of shoulder C4.

TRUNK SYNDROMES

Resemble peripheral lesions.

(1) Upper secondary trunk—Musculocutaneous and external head median.

(2) Posterior secondary trunk—Circumflex and musculospiral.

(3) Lower secondary trunk—Ulnar, inner head of median, internal and lesser internal cutaneous.

External head of median: complete paralysis of pronator radii teres and flexor carpi radialis; weakening of flexor and opponens pollicis. Inner head median: paralysis flexores digitorum and partial of flexor and opponens pollicis and preservation pronator radii teres and flexor carpi radialis.

Associated ischemic paralysis from vascular lesions is common in trunk syndromes.

ISCHEMIC PARALYSIS OF UPPER LIMB

Causes.—Obliteration or ligature of large vessels, tight plaster casts being commonest cause. Not all cases suffer; collateral circulation usually compensates. Nerve injuries alone may cause ischemia.

Symptoms.—(1) Edematous infiltration of all parts, limitation of movement, diminution of all forms of sensation, numbness and burning pains. Skin red or cyanotic. (2) Fibrous transformation of all parts, skin thin, adherent; almost total loss of mobility, hand distended, fingers flexed or curved and thin;

sensation first disappears in hand and gradually diminishes toward root of arm.

Diagnosis.—(1) No peripheral topography to motor and sensory disturbances, but segmentary. (2) Woody, puffy consistence of all tissues. (3) Suppression of radial pulse. (4) No real paralysis but fibrous immobilization of muscles. No polar change, but hypoexcitability.

Treatment.—Unfavorable. Persist in hot baths, massage, mobilization, and galvanic and faradic currents.

SCIATIC NERVE

Lumbosacral Cord.—4 and 5L and 1, 2, 3S. Divides at popliteal space or higher into internal and external popliteal. Branches of sciatic: N. to semitendinosus, long head of biceps, semimembranosus, short head of biceps and abductor magnus—all flexors of the leg.

External Popliteal. — Extensor of foot. Branches: Sensory branches to external and posterior surface of leg and heel; peroneal or external cutaneous to antero-external part of upper leg.

(1) *Anterior Tibial.*—Nerve to tibialis anticus, extensor communis digitorum, extensor proprius hallucis and extensor brevis digitorum. Sensory branch merges with musculocutaneous, supplies dorsal surface of first metatarsal and big toe and especially *first metatarsal space.*

(2) *Musculo-cutaneaus.*— Peroneus longus and brevis and cutaneous branches to antero-external region of leg. Terminals: Internal and middle dorsal cutaneous to external collateral of big toe and second and third toes up to second phalanx.

Internal popliteal changes name to posterior tibial at arch of soleus, and in internal malleolar groove divides into internal and external plantar. Branches of internal popliteal: N. to two heads of gastrocnemius. N. to soleus, plantaris, popliteus, and articular to knee.

Sensory.—External saphenous runs under external malleolus, supplies lower external part of leg and dorsum of little toe and half of fourth toe.

Posterior tibial supplies.—Tibialis posticus, flexor proprius hallucis and flexor communis digitorum. Terminal branches are:

(1) *Internal plantar* (Median).—Abductor hallucis, flexor brevis hallucis, flexor brevis digitorum and accessorius.

Sensory. — Plantar cutaneous to whole inner side of sole and digital cutaneous to big toe, second, third and half of fourth toes and dorsal surfaces of ungual phalanges.

(2) *External plantar* (Ulnar).— Abductor minimi digiti, flexor brevis minimi digiti; adductor transversus and obliquus and plantar and dorsal interossei. Sensory to little and half of fourth toe and dorsal ungual phalanx.

PARALYSIS OF EXTERNAL POPLITEAL

Loss of dorsal flexion of foot and toes, internal and external rotation and abduction of external border of foot; falling of arch. Drop foot and steppage gait. Toes are flexed. Loss of adduction often is compensated for by tibialis positicus (internal popliteal).

Sensory. — Anesthesia of antero - external and posterior surface of leg and dorsum of foot up to ungual phalanx. Usually it is restricted to external surface and dorsum.

Trophic and vasomotor.—Uusually slight. May be edema and cyanosis, desquamation, hypertrichosis and soft tumor on dorsum of foot.

CLINICAL FORMS

May be complete interruption, compression, regeneration and irritation. The latter is uncommon, as external popliteal is not much of a sensory nerve. May be dissociated: anterior fibers for anterior tibial and posterior fibers for musculocutaneous.

II. PARALYSIS OF ANTERIOR TIBIAL

Only the extensor group and tibialis anticus are paralyzed, if low enough only extensor proprius hallucis may be affected. Foot drop; steppage gait.

Sensory.—Small area between big and second toe and dorsum of first metatarsal space.

III. PARALYSIS OF MUSCULO-CUTANEOUS

Loss of abduction, external rotation and elevation of foot outwards; hence, talipes varus. *Sensory.*—Almost whole dorsum of foot.

PARALYSIS OF INTERNAL POPLITEAL

Loss of plantar flexion of heel and toes; collapse of internal side of arch of foot (tibialis posticus), loss of abduction and adduction of toes. Patient cannot raise heel from ground. Splay-foot. After a time there is dorsal extension of foot and toes. Loss of plantar and Achilles reflex.

Sensory.—Anesthesia of plantar surface of foot, back of leg to middle third; external part of dorsum of foot (External saphenous), and ungual phalanges.

Trophic changes are not common in compression or interruption but in neuritic type. Chilblains, mechanical ulcers, and nail changes may occur.

Clinical Types.—All types. Neuritic more common than in external popliteal. May be slight and give a pes equinus. Grave type is frequent. Violent pain on pressure, especially on calf. Marked skin changes, clawing of nails and fibrosis. Forced flexion of foot and fibrosis of plantar tendons and aponeurosis and Achilles tendon. Causalgia is next in frequency to median.

Fascicular topography.—From within outwards; external saphenous, plantar nerves, gastrocnemius, tibialis posticus, flexor longus digitorum and external plantar.

PARALYSIS OF POSTERIOR TIBIALIS

Frequently overlooked; may be limited to plantar muscles and partial anesthesia of sole of foot. Foot is hollowed out and atrophied. Foot griffe. First phalanx markedly extended and last two flexed. Loss of abduction and adduction of toes. Same trophic changes as in internal popliteal.

EXTERNAL SAPHENOUS NERVE

Anesthesia of external malleolus, external surface of heel and foot. May be painful so that the patient dares not walk on heel, and steps on the inner side of the foot.

PARALYSIS OF SCIATIC TRUNK

Complete paralysis of leg and foot muscles and atrophy. Steppage gait. Posterior muscles of thigh often atrophied. Semi-tendinosus usually escapes; hence, flexion of leg is retained. All syndromes may be present. Neuritic type is very common; dry or sweaty skin, contraction of tendons, pes equinus. Dissociated lesions are very common, particularly of internal popliteal alone or external popliteal alone; or one part of nerve may show regeneration and the other not, or interruption in one branch and irritation in the other.

DIAGNOSIS OF PARALYSIS OF SCIATIC

Hysterical or functional disease after regeneration. Actual destruction of muscles may simulate nerve paralysis. Electrical reactions help to decide. Peripheral neuritis may be limited to sciatic or even one branch. Diphtheria, dysentery, frost-bite and asphyxiating gases should be considered. Frost-bite neuritis is painful and may give contractures. All neuritides usually are bilateral. Tibialis anticus frequently escape, like supinator longus in saturnine musculospiral paralysis. History of case is important. Lesion of sacral plexus may cause trouble in diagnosis. Lumbo-sacral hematomyelia shows motor and sensory root distribution and sensory dissociation. Ischemic paralysis shows segmentary paralysis of foot and sensory disturbances, disminishing as we go up, suppression of arterial pulse and presence of some voluntary contraction.

TREATMENT OF PARALYSIS OF SCIATIC

Overcome steppage by means of surgical boots or contrivances with spring or elastic straps attached to tip of shoe and supported on leg, thigh, or shoulder.

SMALL SCIATIC NERVE

First, second and third S. roots. Wholly sensory. Supplies lower part of buttocks, narrow strip on posterior part of thigh down to middle of leg. Lesion shows only anesthesia. Supplies also perineum and scrotum.

ANTERIOR CRURAL NERVE

Second L. mainly third and fourth L. roots. Supplies psoas, iliacus, pectineus and sartorius together with sensory branches to antero-external part of thigh and knee; then motor branch to quadriceps, and internal saphenous nerve to inner side of knee, whole inner side of leg, malleolus and inner side of foot to base of first metatarsal.

Paralysis is rare, generally the result of pelvic injuries, and involves pectineus, sartorius and quadriceps. Extension of knee is lost, except that gracilis and tensor fasciae femoris give pseudo-extension. In walking, patient keeps leg hyperextended, but it caves in on slight flexion. Walking backwards is easy. Hydrarthrosis of the knee is common.

Sensory.—Anterior part of thigh and inner side of leg.

INTERNAL SAPHENOUS

Anesthesia of antero-internal surface of knee, inner side of leg and foot. May be the seat of neuralgia or even causalgia.

Diagnosis of anterior crural paralysis from functional paralysis, muscle injuries in fractures of the femur and root lesions or lumbar hematomyelia.

OBTURATOR

Second, third, fourth L. roots. Supplies, in pelvis, obturator internus. Superficial branch: Gracilis, adductor longus, adductor brevis. Deep branch: Adductor magnus. Function is to adduct thigh; secondarily, rotate outwards and flex thigh. Adduction is not completely lost, as adductor longus is also supplied by anterior crural and adductor magnus by sciatic.

Sensory.—A triangular area on inner middle surface of thigh. Paralysis is rare, as nerve is well protected.

EXTERNAL CUTANEOUS NERVE OF THIGH

Second, third, L. roots. Exit in notch between anterior superior and anterior inferior iliac spines. Supplies external part of buttock (and external part of thigh). Special neuralgia described by Roth is known as meralgia paresthetica and is shown by pain on walking. Causalgia may occur. Meralgic pain is induced by contraction of tensor fasciae femoris.

GENITO-CRURAL

Second L. root. Emerges under Poupart's ligament. Supplies area over Scarpa's triangle, the inguinal supplying the root and inner side of the scrotum.

ILIO-HYPOGASTRIC AND ILIO-INGUINAL

First L. root, similar to an intercostal nerve. Supply motor twigs to internal oblique and transversalis and sensory to upper and outer side of the buttock, to skin over inguinal canal and to inner side of it. This makes a narrow horseshoe on outer, upper and inner side of thigh.

LUMBO-SACRAL PLEXUS

RADICULAR SYNDROME OF LUMBO-SACRAL PLEXUS

Lesions of cauda. Usually dissociated paralyses as distinct from peripheral nerve lesion. Incomplete paralysis unless several roots are injured. May be sensory alone or motor alone. Sensory changes are in longitudinal tracts in case of sacral and oblique in L.

LUMBAR ROOTS

Paralysis of first and second L. gives weakness of qudratus lumborum, transversalis, and anterior muscles of thigh.

Sensory.—Anesthesia of outer surface of buttock and oblique, upper anterior surface and outer surface of thigh.

Third, fourth and part of fifth L. supply all anterior muscles of thigh; crural triceps, pectineus, sartorius (anterior crural); adductors, gracilis (obturator). The thigh appears strangled in the middle. The muscles weakened are: glutei, tensor fasciae, posterior muscles of thigh and leg, especially tibialis anticus (fifth L.), extensor communis digitorum and proprius hallucis and inner head of gastrocnemius.

Sensory.—Obliquely outer surface of thigh, and surface of knee and inner surface of leg and foot. Patellar reflex (third and fourth L.) is absent.

SACRAL ROOTS

In lesions of first and second S., all the muscles of the leg are affected, except tibialis anticus and partly extensor of big toe and inner head of gastrocnemius.

Sensory.—Broad tract on posterior surface of buttock and going down the thigh, outer side of knee and leg, anterior surface of leg and both plantar and dorsal surface of foot to first intermetatarsal space. The Achilles reflex (first and second S.) is absent. Third S. corresponds to inner side of buttock and

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APHASIA AND ASSOCIATED SPEECH PROBLEMS

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The problem of speech from a medical and purely psychological standpoint is so unsettled that contributions to our knowledge, or aids to the understanding of that knowledge which we already possess concerning speech, are surely to be welcomed. Particularly are the difficult problems of speech disturbances in the various types of aphasia in a chaotic state, nor do we have precise knowledge of the mechanisms which cause disturbances of speech in the various so-called functional conditions as in hysteria, dementia precox and manic depressive insanity, etc. Indeed, the speech disturbances seen in dementia precox and general paresis, both organic diseases of the central nervous system, are not entirely clear as to the mechanism of their causation. Further, it has always been an interesting problem to explain precisely the cause of speech defects occurring in Friedreich's ataxia, paralysis agitans, multiple sclerosis, cerebellar ataxias, the various cerebral and cerebrocerebellar diaplegias, and finally, but not of least interest, speech defects occurring under extraordinary conditions of fatigue, stress or emotion. The orthodox views on the development of speech and the localization of the various speech centers are faithfully presented. In them one searches in vain for a clear understanding of normal or defective speech. The influence of the cerebellum in connection with speech has been almost entirely neglected. In these pages an attempt is made to ascribe to the cerebellum an important place in the exhibition of that exclusively human function.

CHAPTER I

At the outset we desire to have it understood that our contribution is based in no instance on any original anatomical investigation, either of our own or other material. In all fairness, it must be conceded that anatomy, both experimental, normal and pathological, has failed to clear up the problems of speech in the various types of aphasia, for instance. It has appeared to the author that a revision of our conception of aphasia is important and, therefore, this clinical study was undertaken. My inspiration for undertaking this work was received from Professor Frederick Tilney, and some of the clinical cases used in this study were observed with his aid and guidance. Others of the cases are from my private practice and from the neurological wards of the Kings County Hospital. The chapter which represents the generally accepted orthodox views on speech is made possible by a translation of Emil Villiger's monograph, "*Sprachentwicklung und Sprachstörungen beim Kinde—Unter Berücksichtigung Hirnanatomischer Grundlagen* (1911)" on speech disturbances and speech development in the child. For this translation I am greatly indebted to Dr. I. S. Wechsler, Instructor in the Department of Nervous Diseases, Columbia University.

After presenting in the first chapter the views of Villiger in abstract, the attempt will be made, by reference to Moutier's figures as the pathological basis and to Marie's argument for a revision of the question of aphasia which appeared in his writing of 1906, to break down the conception of aphasia as it is now usually understood. In doing this, certain references will be made to clinical material of my own and others to show the absolute necessity of such a revision. Following this, our conceptions of speech will be laid down in very broad viewpoints which will be upheld in so far as clinical material can be made to do so by reference to specific cases observed by the author. Clinical studies will be advanced in illustration of theoretical conceptions. Anatomy and pathology having failed to elucidate the problem, we shall in these pages attempt to call clinical medicine to our aid.

VILLIGER'S CONCEPTION OF SPEECH DEVELOPMENT

The first sound made by the human being is a reflex cry, brought about by the need for respiration in the child. Soon, however, the child seeks to give tangible expression to its needs, likes and dislikes; sensations of cold and of being wet, of skin irritation, of hunger and thirst are now often accompanied by crying. Even pleasurable sensations are accompanied by crying, and in the beginning none of these cries can be with any certainty differentiated. At the end of several weeks, however, a specialization of the various cries occurs and there can be distinguished two groups, those cries which indicate a state of well being and those which indicate the contrary. States of well being are characterized by a cry which is associated with smacking, sucking and "lalling." The lalling activity of which Villiger speaks consists in the production of sounds, syllables and multiples of syllables. Vowels are combined with consonants in a lall monologue. According to Villiger, the first sounds produced are not always those which require the simplest physiological strain, for gutturals are very often pronounced before the labial sounds. Many exceptions to the general rule, however, regarding this is possible, because the environmental influence surrounding the child, particularly the amount of care and attention a child receives from its parents or relatives, will determine the speed and character which speech development shows in the individual child. There are two periods in the child's development when these undifferentiated sounds are made. The first one occurs in most children between the ages of three to six months and is characterized by the formation of laryngeal and palatal sounds in combination with Ah, Ay and Oh, besides many inarticulate sounds. The second period occurs between the ages of six months and one year, a time during which we have the production of tongue, lip and palatal sounds, the first two predominating. In the first period, the child does not initiate these sounds with any purpose but amuses itself in the production of them. The frequent repetition of sounds in the first lalling period is explained by the structure of the articulating organs and the manner of sound production. The air current in passing the larynx and the mouth is impeded and made into tones by the speech organs. As the air first passes by the palate, the palatal sounds are the first to be formed. The exercise which the lips get in sucking conditions the early production of labials and dentals. Every child is born with a psycho-motor tendency or instinct to speech, and irrespective of any sound stimuli in the environment, all children will seek expression in speech.

The child is a motor being, much more so than the adult. In play, it gives expression to all its inner feelings and thus the speech organs in the beginning are used for this purpose; later, all the other organs of the body over which the child exercises any control are used. For unpleasant emotions there is the cry, and soon pleasant emotions are differentiated through another form of sound production which Villiger designates as lalling. After the first period of lalling, namely, from three to six months, a new factor enters to shape the development of speech, which is hearing. In the first weeks of babyhood there is deafness. Soon, however, it shows the ability of hearing by blinking or shrinking in fear at sudden sounds. At the third month a child will turn its eyes and ears in the direction of sound and will be attentive and seek eagerly for acoustic impressions. Hearing is much more important for the development of speech than seeing, not only for speech but also for the psychic development of the child as a whole. The loss of hearing robs the child of any approach to its soul. As the child learns to hear it begins to pay attention to its own lalling and noise production, it soon begets a correct interpretation of its own speech sounds and gains control over the musculature which produces them. Speech sounds which the child itself makes and the sounds which it hears are the basis of speech. He first hears what he himself speaks and then speaks what he hears from others. From this spring imitation and repetition. At first there is self imitation and then there is gradually added to this, imitation of speech which is heard or of other natural sounds. Repetition such as we understand it in the adult appears much later than this stage of development in the child. Other senses enter into this process of imitation of speech and can be seen and even felt by the child, but nevertheless, hearing is much more important than any of the other senses for the development of speech. This is illustrated by the case of deaf-mutes. However, by watching the lips and the mouth, the child (as may the adult) learns to understand speech. Helen Keller can understand speech by holding her hand on a speaking person's larynx. Therefore, she employs the sense of touch in the understanding of speech. But we soon notice that children mutilate speech in imitating it, either because of lack of attention, difficulty in hearing or inability to repeat difficult sound combinations or even possibly because of indistinct speech on the part of adults who speak to the child. Further, this mutilation of speech in imitation may be due to the fact that the child repeats sounds which he hears which are not speech. The first imitations are altogether voluntary. Actual imitation comes much later in children than is generally supposed. Stern found that this ability in children to imitate shows itself at nine months, but mostly is limited to gestures, unarticulated sounds and to the cadence of voice. Repetition of articulated sounds develops with a bound and in the third year this is quite prominent. Indeed, words are now understood and a few are spoken with understanding. In the course of learning speech, there is often a peculiar period of echolalia.

As soon as the child learns to lall, another factor enters. The adult who listens learns to understand some of the words from the babbling of the infant and repeats them to the child, singly or in sentences. These combinations picked by the adult from the child's lalling are in turn repeated by the child and meaning may be attached to them by both. This adult-child language varies with the mentality of the infant, but often children will show some discrimination in choosing from the language which is offered them by the adult. This discrimination is determined by the fact that it chooses the sounds which are phonetically most easy for it to produce. Other factors may influence this choice; namely, the surroundings and the lines of interest which the child may be trained to show in its selection. To the theory that children can invent words, Villiger answers that children have often been observed to speak words which have never been spoken to them by adults. This, however, he explains by the fact that infants mutilate words and in the beginning do not hear properly; or meaningless, badly pronounced words are spoken to them by adults, and thus the nature and quality of its speech and sound intake are not properly controlled or standardized, and in this way the child will employ words for things other than those for which they are intended. Again, even adults will attribute meaning to senseless articulations and will often call them words when it is perfectly obvious that the child has absolutely no meaning to convey by the sound produced. Speech understanding is of great importance for the production of speech imitation. Very early one can observe the impressions which are made on the child's feelings by the mother's voice or other signs. At first the child's feelings may not show a differentiation in reaction to what is spoken to it. Later we notice how the child's feelings are expressed in various movements as shown when certain things or persons are mentioned, the infant responding to these words by turning the head or the eyes in the direction of the objects mentioned. For instance, on saying "tick, tick," and holding a watch before the child, it learns to gaze in the direction of the watch, and even if the words "tick, tick," are said and no watch is immediately visible, the child will look in the direction from which the sound is made. The child cannot be said at this stage to have a visual conception of the object. These traces of speech understanding appear more clearly in attempts at training the child. Thus, when a child is taught to stretch out its hand upon "Give me your hand," it learns to associate action with sound. This, however, is really not actual speech understanding but only a mechanical repetition of practiced action.

Soon the child learns to attach great importance to speech. He sees in it a close connection with objects and occurrences and finds that it is the only means for communication and discrimination. In time he learns to recognize words as signs for objects and finds understanding is also furthered by gestures. Thus mimicry acts as a connecting link between sound complexes and expressions. Finally, speech is understood without the accompanying mimicry or gesture. In many cases speech understanding may be far in advance of actual speech, for normally there exists in all children a period when a great deal of what is said is understood but only a very small proportion of this can be uttered by the child. This is designated by Villiger as hearing mutism and might be compared to what we see in the condition known as motor aphasia, or in some types of psycho-motor retardation such as we see in depressions.

According to the psychologist Neumann, the first words which a child utters give expression to emotions and desires. Following this, the volitional stage ensues, finally the intellectual stage of speech is developed, and in this stage objectivity is attached to words and they are thus used for designations and communication. Only in much older children are words used associatively, for a child may employ one word for a number of objects or events, simply because the word was first used in a certain way, the child unthinkingly connecting it with all things that bear a resemblance to that particular instance. The child may recognize and designate objects by signs or by combining parts, and may employ one of these parts for the whole object. For instance, it may call all quadrupeds "Bow-wow" or dog, and all women "Grandma." The reason for this lack of associativeness is the fact that there is still an incomplete perception of things. This lack of logical conception of words as symbols may be due to want of attention, lack of power of memory and narrowness of consciousness. Gradually, however, the child learns to know that separate word symbols are used for different objects and by means of constant questions and answers and corrections acquires the proper use of these words and various conceptions from which grow systematic and conscious ability of speech. It is fairly well established, first, that there are no sharp lines of demarcation between the various stages of speech development. Secondly, that the development persists at varying degrees of speech, and thirdly, that speech development and the development of intelligence usually have nothing to do with each other. This is almost a literal translation of Villiger's view, shared by a great many, that speech and intelligence may be entirely separate things in the development of the individual. How much we disagree with this conception will be brought out later. That this is a fundamentally important point is obvious, for if this statement is true then the anatomical localization of various speech centers is justifiable, and the conceptions which have allowed so many to place diagrammatically limited centers over various speech functions in the brain are correct. However, we hope to be able to prove that this statement is not logical, and is, therefore, not true and that the development of speech and intelligence go hand in hand and are interdependent to such an extent that they may be, to all intents and purposes, one and the same process. Villiger further states that normally articulated speech should be developed by the eighteenth month and that it should be a fully developed function by the end of the fourth year. Normal variations may, however, place the outside limit up to six or seven years, particularly if one considers sentence construction and stylistic expression to be part of speech development. Indeed, after this, cultivated refinements of speech are included within speech development and may be said to continue throughout the school period.

The building up of the speech mechanism furnishes the second part of Villiger's interesting little book. The first part of this deals with a review of the evolution of the conception of psychic functions and their localization in the brain. He calls attention to the work of Gall in 1820 as showing that the cerebral cortex was the seat of psychic functions. Flourens and Foville established this conception as a fact by experimental and pathological work in 1830. Marc Dax in 1836 observed speech disturbances in patients with right hemiplegia. His son, G. Dax, in 1863 showed that the lesion in such cases was in the left hemisphere; and, working independently, P. Broca, in the same year, showed by a report of his first two cases that the localization of the seat for articulated speech was in the foot of the left third frontal convolution. He further describes how, as a result of pathological research and histological differentiation of the various layers of the cortex, it was determined that different cortical regions had specific functions. He describes the failure of observers, particularly Beaunis and Fournier in France, and Nothnagel in Germany, to localize centers in the cortex by injecting liquids. The oft-quoted experiments of Hitzig and Fritsch in 1870 proved by electrical stimulation the presence of motor areas in the cortex of animals.

Ferrier, working with the faradic current in 1872, confirmed their findings. Sherrington and Greenbaum in 1901–02 confirmed this work on apes, and finally F. Krause added his confirmation, working on human beings. References are made to the work of numerous physiologists, such as Monk, Nothnagel, Corville and Goltz, which definitely showed that motor or sensory activities disappear on destruction of definite parts of the cortex. Attention is called also to the clinical work of Charcot, Hitzig, Kussmaul, Wernicke and Dejerine which confirm the experimental work of the others. From all this, Villiger decides that it has been established that the cortex consists of physiologically differentiated sections in the sense that definite portions have specific elementary functions. This conception belongs particularly to Wernicke and is reenforced by the work of Berlin in 1858, Arndt in 1867, and Meynert in 1886–72, who showed differences in the structure of various convolutions, and of W. Betz in 1874, who showed the presence of the giant pyramidal cells in the ascending frontal convolutions. Following these, the researches of Bevan and Lewis, Clarke, Obersteiner, Golgi, Ramon Y. Cajal, etc., first showed that the cortex in imbeciles and idiots lacked functionally active nerve cells, establishing thereby the conception that nerve cells are essential in psychic activity. Gennari, Soemmering, Vicq-d'Azyr, Rennak, etc., showed the presence of fibers, both projectile and association, in the cortex, and Tutzek and Zacher observed the disappearance of these fibers in general paresis and senile dementia. Vulpius and Kaes studied the development of fibers at various ages. Flechsig showed that fibers and tracts, both in the cortex and in the cord, become myelinated at different periods of development. By means of the special methods discovered by Golgi, it was observed that fiber and cell are one structure. Cajal gave exact description of the motor and sensory cortical layers and Broadman worked out the typographical localization. The keen and masterly work of Hughlings-Jackson is not mentioned with those of Charcot, Hitzig, Wernicke, Dejerine, etc., as the clinicians who helped in the localization of cortical function. Nevertheless, Head regarded Jackson's work of such great importance that he undertook to gather and edit a number of his publications which appeared together in an issue of Head's periodical, *Brain*, parts 1 and 2, Vol. XXXVIII. Further reference to this work will be made later.

To continue with Villiger's conception of speech, we find that in the third or fourth month of embryonal life the cortical cells are undifferentiated. Toward the last quarter of fetal life, one can distinguish six layers. In the interval between these two periods there occurs a rearrangement of cells into layers of lesser or greater density with differentiation in cell structure, together with a regrouping of fibers. The further development of this histological localization continues in the child up to the second or third year and one may assume a parallelism between organic and psychic development from these facts. This may be borne out by the observation of H. Vogt, who showed that there was no structural differentiation in idiots. This has been shown further phylogenetically, for the same cortical area differs in different animals during the various stages of development. The lower the animal, the more granular cells there are; the higher the animal is, the more pyramidal cells are found. According to Romdoni, those layers in the human which are developed earliest show a preponderance of granular cells, and those which develop latest, a preponderance of pyramidal cells. Further, there arises an association between the various cells in the same cortical area, as well as between different cortical areas. From which may be adduced that while for more limited functions there are distinct cortical fields, for higher performances a great part, if not the whole, of the cortical area is necessary. Because of this very point do we differ from Villiger's conception that speech and intelligence develop separately, for the remarks just recorded from Villiger's pen are susceptible of being called upon to prove that speech is merely the visible expression of intelligence and develops apace with the latter. Villiger says that the following main centers are distinguished: First, the motor center which embraces the central convolutions, particularly the precentral, the posterior part of the frontal lobe and a large part of the lobulus paracentralis. Second, sensory centers; (a) touch, pain, and temperature in the post-central convolution and the adjourning part of the parietal lobe, possibly also the precentral convolution. In this region is found sense of position and movement, of space and place; (b) hearing in the middle and lower part of the temporal convolution and particularly the convolution of the insular lobe; (c) a visual center in the occipital lobe, particularly the cortex of the calcarine fissure; (d) a small center in the front part of the gyrus hippocampus and cornu ammonis which are for taste and smell—the outer part probably for taste and the inner part for smell.

These motor and sensory areas occupy perhaps a third of the brain cortex, the rest of it being considered by Flechsig as areas for association centers which are the seat of higher psychic functions. This latter author explains the development of centers on the ground of myelinization as follows: At birth only smell and taste centers are developed, then come in order, myelinization and function to the centers for touch, vision and hearing. Later, the individual territories of the psychic centers. According to this author also, the projection areas, motor and sensory, are the only ones in touch with the lower brain centers, whereas association centers are connected only with one another and with the projection centers. The cytological studies of Broadman have shown histologically different areas in the so-called association centers. While little is known of the function of the anatomically circumscribed areas, the existence of memory centers seems certain. Thus, there are lesions in centers of perception which destroy perceptions but not memory pictures, and on the other hand, there are lesions near perception centers such as the visual or auditory, where the memory picture may be lost but not the actual perception. For instance, we may have psychic blindness wherein the patient knows the form and color of an object but cannot tell what the object is, or in psychic deafness, the speech may be perceived as sound, but not recognized as a symbol. Or again we may have tactile agnosia wherein objects are felt and perceived through the sense of touch but not recognized.

Speech centers embrace a definite cortical area of the left hemisphere, according to Villiger, wherein he follows a great number of authors on this subject. These are placed (a) at the foot of the lower frontal convolution (Broca's area) which possibly impinges on the lowest portion of the precentral convolution and the insula. Injury to this area leads to loss of voluntary speech, repetition and also reading aloud. This statement may be read again and again in various works on this subject despite the fact that it has been

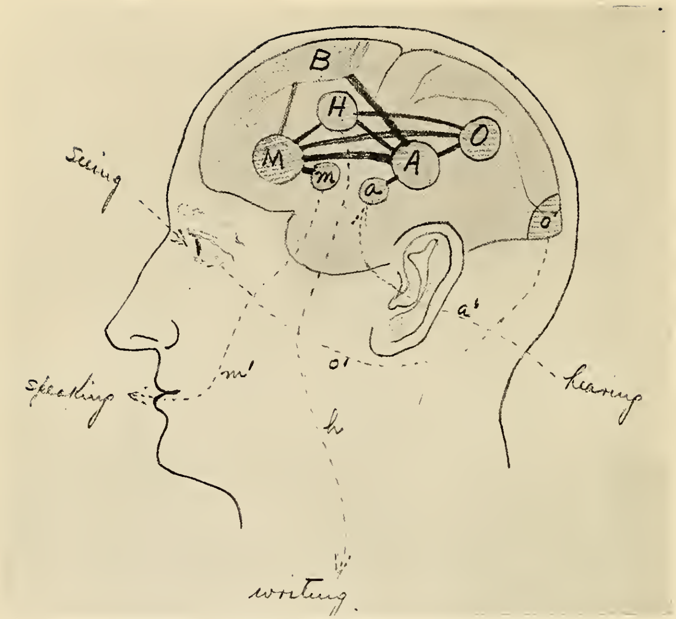

Fig. 1. Diagram of Sound Language and Written Language

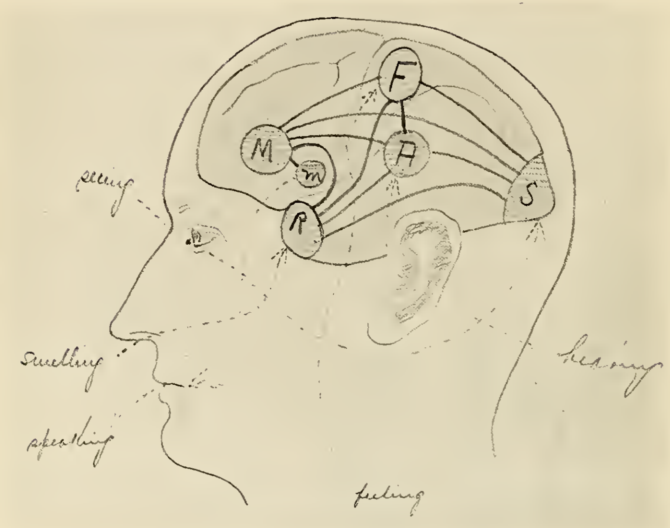

Fig. 2. Diagram of Concept Formation

proven time and again that lesions in Broca's area have been observed at autopsy without any speech disturbance whatsoever. (b) In the posterior third of the superior temporal convolution. Next to the lower parietal lobule lies the sound picture or auditory center. This is the Wernicke center which contains the memory pictures of heard and spoken words. It is the center which is injured when word deafness occurs, that is, such a patient hears words but lacks understanding. (c) In the angular gyrus lies the visual center for memory pictures of written signs. Alexia occurs when this center is injured. In other words, we have word blindness. (d) Some consider the existence of a separate writing center in the foot of the middle frontal gyrus, but it is likely that this belongs to the motor center for the hand.

All these centers are memory centers for the representation of movements, of articulation, of acoustic and visual pictures and of signs of speech.

Figure 1 shows the connections between the motor and the sensory speech areas. When a word is spoken it reaches through the path *a′* and a memory picture for recollection or recognition is formed in the sensory speech area *A*. From here the impulse travels to the motor area which enables one to repeat the word. But as the child's musculature is unskilled and its attention restricted, the repetition at first is incorrect. Gradually, a memory picture for the expression of the word is formed and deposited in a definite area *M*, which becomes the motor speech area, so that later, in the repetition, the sensory impulses from *A* travel to the motor speech area *M*, and the memory pictures from this center set in motion the necessary coördinated movements in the motor center *m* in the lower precentral convolution, and from here this impulse travels to the speech musculature by way of the speech path *m′*. The process, then, is *a′-a-A-M-m-m′*.

Now we can explain the concept formation in the child during speech development. Suppose the child sees a rose. From the eye an impulse travels to the visual cortical area where the rose is seen. Immediately a memory picture is deposited in the center *S* (Fig. 2), so that the child can recall form and color. As the child can also smell and feel the rose, memory pictures are deposited respectively in *R* and *F*. As these centers are connected by association fibers, upon seeing the rose, there are awakened in the child memory pictures of its smell and the feel of the petals; thus a concept "Rose" is formed out of partial representations. As the word "Rose" is spoken to the child an acoustic memory picture is deposited in *A*, so that the child recognizes the word as a sign for the flower. A composite representation is the result.

For the understanding, it is necessary that the speech center *A* enter into closer connection with those centers whose association makes possible the formation of a concept. To represent this graphically, it is necessary to inscribe besides *A* in Figure 1, the memory centers *S*, *R* and *F*, and their interconnections with the sensory speech center *A*. It is also necessary to insert a single "concept" center *B*. But it must be emphasized that concept formation is not to be represented as a simple event but one which is complex and depends on the integrity of numerous associated cortical areas.

Through the connection of the sensory speech center with the concept center, the foundation is laid for the possibility of speech understanding. Not only can the child, at this time, speak without understanding (*a′-a-A-M-m-m′*), but it can understand without speaking, (*a′-a-A-B*). From this is derived loud repetition of words with understanding—actual speech. At first, this follows on the path *B-A-M-m-m′*, and then, in consequence of late associations *M-B*, on the path *B-M-m-m′*.

Another step is needed for the understanding of speech disturbances. The first associations represent speech in the broad sense. This is broadened out by the hearing of written speech, reading and writing. The word signs are to be looked upon as signs of sounds and not concepts. Individual words are decomposed into letters and syllables and an optical letter-picture is associated with each sound, vowel or consonant. In Figure 1, *O* represents this memory center for written signs to which impulses from the eye travel by way of *o* to the visual center *O*. As the child learns to read aloud, a picture of the letter is deposited in *O*, and as it syllabilizes aloud a sound picture is formed in A and at the same time a memory picture of the necessary coördinated movement of the speech musculature is laid down in M. That is, the optical center O enters into connections with the sensory center A and motor center M. For writing, a further association is necessary of O with the center for the movement of the hand H. At first, the child copies mechanically ($o'\text{-}o\text{-}O\text{-}H\text{-}h$), through which graphic-motor memory pictures are developed in H. Later when the child writes from dictation, it softly repeats to itself each word ($a'\text{-}a\text{-}A\text{-}M\text{-}m\text{-}m'$), wherefore, in consequence of the connections $A\text{-}O$ and $M\text{-}O$, the picture of the letters is awakened in O and the impulses from here to H make possible writing from dictation. Finally, the connection between H and M and A becomes direct and voluntary or conscious writing becomes possible.

From the diagram of sound and writing speech, the following may be derived:

Repetition............. $a'\text{-}a\text{-}A\text{-}M\text{-}m\text{-}m'$
Speech understanding... $a'\text{-}a\text{-}A\text{-}B$
Spontaneous speech..... $B\text{-}A\text{-}M\text{-}m\text{-}m'$ / $B\text{-}M\text{-}m\text{-}m'$
Reading................ $o'\text{-}o\text{-}O\text{-}\underset{(M)}{(A)}\text{-}B$
Reading aloud........ $o'\text{-}o\text{-}O\text{-}\underset{(M)}{(A)}\text{-}B\text{-}M\text{-}m\text{-}m'$
Copying.............. $o'\text{-}o\text{-}O\text{-}H\text{-}h$
Writing from dictation.. $a'\text{-}a\text{-}\underset{(M)}{(A)}\text{-}O\text{-}H\text{-}h$
Spontaneous writing.... $B\text{-}\underset{(M)}{(A)}\text{-}O\text{-}H\text{-}h$ / $B\text{-}M\text{-}H\text{-}h$

Speech Disturbances in the Child

According to the diagram in Figure 1 the speech disturbances may be divided as follows:

It is assumed that the speech mechanism is built out of corticipetal, intercortical and corticifugal pathways. The main inlet is the hearing pathway. As has been shown, visual and touch pathways play a secondary rôle. The trapezoid form in the figure shows the intracortical association of the speech centers to form a speech zone. The speech zone is connected with the concept center. The main outlet is the motor speech pathway from the motor speech center to the speech musculature.

In disturbances of the hearing pathways, speech cannot ordinarily be developed since no center can be developed for the sound of words. The same may be true of a high grade of hardness of hearing. In this case we deal with hearing mutism. Speech is unintelligible even in those who have become deaf in a later period.

In disturbances of the central or intracortical pathways, we have two kinds of central or cortical speech disturbances. First, those disturbances of intelligence in mentally diseased or mental defectives are known as dyslogias or dysphasias. Second, lesions in the sphere of the speech centers are known as aphasias.

Aphasia may be defined as loss of or encroachment upon the ability to change concepts into words, in spite of the integrity of the speech musculature, and a loss of the ability to understand things spoken despite the integrity of the sense of hearing.

Cortical motor aphasia depends on a lesion in the motor speech center M. The patient cannot speak spontaneously and cannot speak words. Reading, spontaneous writing and writing from dictation are also encroached upon as both reading and writing depend upon the integrity of the motor and sensory speech centers. Copying is possible. Cortical sensory aphasia (Wernicke's) is caused by a lesion in the sensory speech center A. Understanding is lost, also repetition, reading and writing from dictation, while spontaneous writing, copying and speaking are retained. The latter shows itself in paraphasia.

Subcortical Aphasias.—Motor subcortical or pure word mutism occurs in a lesion between Broca's center M and the speech muscle center m. Inner speech remains intact; reading, writing and speech understanding are retained, while speaking, repetition and reading aloud are lost. In the case of subcortical sensory aphasia (pure word deafness) which is very rare, the lesion is in the path from a to A. Inner speech is intact; reading and writing are possible, speech understanding is not. Total aphasia means destruction of both centers M and A. Transcortical motor aphasia means a breach between M and conception center B, and shows loss of voluntary speech and writing. Transcortical sensory aphasia means a breach in the path from the sensory center A to the conception center B and shows a loss of understanding of speech and writing. Conduction aphasia means a lesion in the paths between M and A. Repetition is affected while the understanding of speech and writing and copying are possible. Speaking and writing are retained but in the form of paraphasia and paragraphia.

Aphasias of individual senses.—Optical aphasia where the patient cannot tell objects by seeing them but can through the other senses. Alexia is a symptom complex in which the patient is blind to letters and words and may be combined with agraphia when a lesion is in the neighborhood of the optical speech center. Amusia, or inability to sing or comprehend melodies, and amimia, which shows loss of ability to give mimic expression, are due to lesions in the main centripetal path from the motor speech center which leads also to disturbances of articulation: dysarthrias. Such disturbances may be organic or functional and if they are dependent on congenital or acquired defects or lesions of the external organs of articulation and their motor nerves, they are known as dyslabias.

STUTTERING.—Villiger says that stuttering occurs in from 1 to 1½ per cent. of school children and that this defect is peculiarly a disturbance of speech and is seen usually in children who are attending school. He described two types, one in which a tonic spasm of the speech musculature occurs, resulting in an extreme difficulty in initiating the word, beginning with a prolonged pause with stumbling before the word can be started. This initial prolonged stumbling having been overcome, the rest of the word is said quickly; for instance, in pronouncing the word "day" there is a prolonged tonic struggle with the letter *d* followed by a more rapid pronunciation of the rest of the word. The other type of stuttering is caused by clonic spasms in the speech musculature resulting in a frequent repetition of the initial letter or syllable of the word; for instance, if again we use the word "day" as an illustration, the *d* would be repeated half a dozen times in short spasmodic gasp-like sounds. The difficulty seems to be mostly with consonants although vowels at times are also spoken with stumbling. Stutterers can usually whisper or sing without impediment. Numerous associated movements occur in the stutterer during the tonic or clonic movements, such as wrinkling of the muscles of the face, twitching of the extremities of the body, wrinkling of the forehead, blinking, movements of the lower jaw, etc. There may also be disturbances in breathing or swallowing. The mechanism of the causation of this is ascribed by this author to a stage in the development of speech in the child when there is a dissociation between the child's desire to speak and its skill in producing sounds. The child, in attempting to overcome this, often blurts out sounds and repeats them in the attempt to speak quickly. If such a child is not taught to speak slowly, the defect grows until it cannot be removed. Slow speech and repetition in children should not be too hastily condemned, for it may be an attempt on the child's part to speak correctly, and should be encouraged in most cases. It is very important in this stage of speech development not to destroy the child's self-confidence. This period of disproportion between the desire and the ability to speak usually begins about the third or fourth year and grows much worse throughout the school period; or it may be brought out for the first time when the child attends school. Bright children are more apt to be the victims of stuttering because they are conscious of the natural defects of their speech and more sensitive to ridicule. Expressions of amusement or actual ridicule on the part of adults at this time increase the fear and self-conscious feeling in the children and lead to a loss of attention and interest and often to backwardness. Villiger says that masturbation may occur coincidently with stuttering and is injurious to the proper development of speech because the nervous system is unfavorably influenced and feelings of anxiety concerning the habit add to the difficulty. Some children gain stuttering by imitation. This defect may be hereditary and alcohol may predispose. Boys more naturally suffer from it than girls. Injuries to the skull, excitements and infectious diseases predispose. Kussmaul says that stuttering is a spastic coördination neurosis of functional character.

The treatment recommended for overcoming this defect is systematic speech exercises, psychic treatment, removal of anxiety and awakening of self-confidence.

Another form of stuttering for which the proper English equivalent could not be named but which Villiger calls "poltern" is characterized by over-haste and swallowing of words or syllables so that language is unintelligible. This distinction is drawn between stuttering and these cases of "poltern." The defect is overcome if the child's attention is called to it whereas in stuttering it is made worse by this procedure.

Stammering.—This is ascribed to a faulty development of speech during the lalling period when the child is learning imitation. Individual sounds are spoken incorrectly and repeated by the child who mixes some of the sounds which it cannot speak with others or leaves them out entirely. If this defect is not corrected, we have functional stammering which is different from the stammering due to organic disease of the organs of speech; that is, the larynx, palate, pharynx, jaw, teeth, tongue and lips. Villiger insists that stammering is a functional disease and is an arrest of speech development. Stammerers vary usually in their manifestations of speech defect. When pronunciation of the letter *r* is disturbed, it is technically known as rhotacism; when the letter *r* is interchanged with other letters, particularly *l*, *w* and *g*, it is pararhotacism. Faulty construction of *l* is lambdacism, whereas the substitution of *l* for other sounds, usually *d*, *t*, *s*, *j* or *n*, is paralambdacism. Faulty expressions of *s* and its interchange with other sounds are seen exceptionally and are known as sigmatism and parasigmatism and in certain forms as lisping. Difficulty with the expression and interchange of the letters *g* and *k* with *d* and *t* are known as gammacism and paragammacism. To summarize, stammering then is faulty expression. Stuttering consists in getting stuck and halting at certain sounds. A sufferer from stammering speaks fluently but does not express himself correctly. This defect often disappears in the first school years without treatment. A congenital form of stammering is recognized, the cause of which is unknown and is usually associated with other central developmental retardations or defects of intelligence.

Agrammatism.—When the child first learns to speak he, of course, does not speak grammatically, but if this persists after the fourth year, a real speech defect must be said to be present. Such children cannot speak sentences at all and use no inflection or connections; for instance, "soup eat, garden go." This is found in idiots and greatly retarded children, such as deaf mutes or stammerers. The defect may be in optic, acoustic, tactile or motor spheres, or in disturbances of attention and memory. Sense perceptions leave few traces in the memory, and these children show want of skill in the motor system of the body besides the speech musculature. It will be noticed in this connection that Villiger gives no, or only a very meager suspicion, as to the clinical defects from which these children suffer. In other words, we are not told whether the difficulty is in the cerebral motor functions or in the cerebellar motor functions or in both. A mild form of agrammatism wherein the memory and attention are better developed but in which psychic defects can still be said to be present, shows itself in deficient inflection and the absence of syntax, and a peculiar arrangement of words. In still another form, the child can speak spontaneous sentences but one has difficulty in making them out. These patients are likened to foreigners who are learning a strange language. However, they can repeat correctly simple sentences. This type occurs in children between the ages of ten and fifteen years and depends on articulatory disturbances of long duration in part organic and in part functional.

Hearing Mutism.—This may occur despite the presence of normal hearing and normal speech apparatus, together with a sufficiently well developed psychic function. Coën in 1888 called attention to this, although it was formerly known as congenital aphasia and mutism without deafness. This condition depends on a central retardation of development of speech; the pathology and etiology is little known. The presence of neuropathic taint in the families in which this defect occurs is postulated. Alcoholism is again suspected as being a baneful influence in the production of this defect. Adenoids and tonsils may contribute, together with inadequate development of collective motor functions. According to Gutzman, this symptom complex is due to a mal-development of the associative speech mechanisms. It will be recalled that the incidence of imitation shows itself in general movements and in special speech movements. In some children this incident appears rather early, in others late. These last also learn to walk late and are awkward, and often want of skill shows itself also in the movements of the muscles of speech. This want of skill is overcome by constant usage and exercise. Villiger again reminds that understanding produces actual speaking and that the desire to speak may not be in proper relation to the ability of the child to speak and so repeated efforts to deliver words result in failure, the child becomes discouraged and gives up and remains dumb through fear. Hearing mutes are said to be normal mentally, but it has been shown that the intelligence has suffered and that in these cases attention and memory are faulty. Out of Villiger's own words, therefore, we have evidence which would tend to show that the entire development of the speech mechanism goes hand in hand with the development of intelligence in the child and that it may be perfectly possible for their evolution to occur at the same time and may even possibly be an identical process. As will be brought out later, there seems to be abundant evidence to support this view, for in a great number of clinical states wherein speech is affected, there is a corresponding defect in the mentality or intelligence. It will be noticed, too, that those children whom Villiger calls hearing mutes, have speech defects despite the presence of absolutely intact hearing mechanisms which Villiger, together with others, have insisted were the most important pathways for the proper development of the speech function. In some cases, which are more probably cases of deaf-mutism, acoustic defects are observed or visual defects may occur together. For instance, with the eyes shut, these children do not well differentiate sounds which are heard and may not be able to localize them. Pictorial representations and the memory for form and color may also be defective. Spacial representation is faulty and defects may be found in the motor and tactile spheres. Hearing mutes cannot tell with the eyes shut whether things placed in their hands are light or heavy, dull or sharp, and on the motor side they are awkward in general movements, particularly those of the tongue and lips. The memory is poor and they are not able to reproduce from memory movements which have been shown them only a short time previous. It may be relevant here to call attention to the fact that these cases are also called apraxia by some authors. Taste and smell are better preserved. It may be that those cases which Villiger has called hearing mutism correspond to types of ideational and motor apraxia occurring in defective children as we understand them in this country. The author continues by classing these cases of hearing mutism under the heading of aphasia. First, the motor form which is the most common and corresponds to motor aphasias. Such children speak nothing or single words only, but they understand everything. Second, the sensory form corresponds to cortical sensory aphasia. Such children hear but do not understand. This is psychic deafness. Hence, they do not learn to speak although they can imitate a little and repeat. They understand gestures and obey commands. These cases are comparatively rare and may be mistaken for deaf-mutes. A third form with both motor and sensory defects shows itself in children who understand single words but not complete sentences. Children with hearing mutism should be carefully studied so as not to mistake them for deaf-mutes because of the difference in the prognosis. In deaf-mutes the prognosis is not nearly so good as in cases of hearing mutism, who can, with careful training, be greatly benefited.

Congenital Word Blindness.—This consists in the ability to read single letters and figures but the inability to combine them into words, and exists in one out of every 2,000 school children. It is more common in boys. The lesion is placed in the angular gyrus. It is usually not severe and is due to a retarded development of the visual speech centers. The verbal alexia which results is comparable to that found in pathological cases in adults. The condition is first recognized at school and is not detected earlier, because they learn to read from memory and often copy well, but when told to read backward or to write down the names of familiar objects they are unable to do so. Repeatedly, one finds striking forgetfulness, such as the inability to carry out orders for purchases. Here, besides the inability to read words, there is the added inability to read letters which are in a vertical plane; notes made in a continuous horizontal line can be read. Early recognition and persistent teaching may bring about great improvement. It is on record that men born word-blind have composed scientific works, though, to be sure, the manuscripts had to be revised and corrected. These children should not be put in special classes for defectives. Assistance outside of school hours may be given besides the regular school work. Blocks may be employed in the study of writing and reading. Marburg suggests the training of the left hand for writing in order to develop the reading center in the opposite hemisphere.

DEAF-MUTISM AND HARDNESS OF HEARING.—Congenital or acquired deafness or hardness of hearing leads to corresponding deaf-mutism. Deafness acquired before the seventh or eighth year leads to mutism. Boys are more naturally affected. Congenital cases occur more commonly in the female, acquired cases in the male. Hereditary defects affect more the female children, and children's diseases more commonly strike the auditory apparatus of the male. The United States shows fewest and Switzerland the most, deaf-mutes. Mountainous countries where endemic cretinism is common show a great number of deaf-mutes. Jews also show a large proportion. Heredity plays a part, but most often not direct, but collateral, heredity. Several children of one family are often affected. Consanguinity may be a cause. Alcoholism, syphilis, mental diseases, weak-mindedness and squint or weakness of sight in the ascendants are said to be causes. Scarlet fever and meningitis account for most of the cases of acquired deaf-mutism. Other infectious diseases, otitis media, or fracture of the skull are responsible for individual cases. Those diseases which affect hearing usually occur during the first or second year. In the congenital form only a few pathological changes have been shown in the cochlea, while in the acquired forms distinct disturbances have been observed. Deaf-mutes also show other bodily defects; mental defectiveness and retinitis pigmentosa have also been found. Bezold has shown that in many congenital and in some acquired, forms of disfunction there is still some hearing left. He showed that only a limited part of the scale is necessary for hearing speech (*B—G*). On enunciating clearly and loudly directly into the ear, he found that deaf-mutes can learn to speak; in addition to this, he has the patient watch the mouth. The better to do this, a mirror is placed in front of the deaf-mute and he is asked to observe the movements of the lips of the teacher as he speaks into the patient's ear. Children who have learned to speak but have become deaf before the age of seven, may become deaf-mutes. Deafness or hardness of hearing acquired after the age of seven leads to unclear and monotonous speech. Such children learn to depend more and more on signs and gestures and their vocabulary becomes limited. It has also been shown that in high grades of deafness, there is an accompanying mental retardation. On the other hand, in mental defectives who are also hard of hearing, it is difficult to say whether the mental defectiveness or the hardness of hearing is the cause of the mental retardation.

CHAPTER II

Lest it should seem that the abstract of Villiger's work already given has been in any way a definite statement entirely or even partially satisfactory regarding speech disturbances or speech development, we must proceed to show the fallacy of such a belief.

Head[9] starts his introduction to Hughlings-Jackson's work on aphasia and affections of speech by saying:

"It is generally conceded that the views on aphasia and analogous disturbances of speech found in the text-books of to-day are of little help in understanding an actual case of disease. The tendency to appear exact by disregarding the complexity of the factors is an old failing in medical history. Each patient with a speech defect of cerebral origin is stretched on the procrustean bed of some theoretical scheme: something is lopped away at one part, something added at another, until the phenomena are said to correspond to some diagrammatic conception, which never has, and never could have existed. And yet neurologists continue to cling to these schemes, modifying them to suit each case, conscious that they do not correspond in any way to the facts they are supposed to explain."

The dissatisfaction which Head shows by this statement concerning the type of conceptions of aphasia and speech disturbances as described by Villiger was shown even more forcibly by Pierre Marie[22] who in 1906 set about entirely rearranging our conceptions of this subject.

Before this, Hughlings-Jackson[15] had stated that "whilst I believe that the hinder part of the third left frontal convolution is the part most often damaged, I do not localize speech in any such small part of the brain. To locate the damage which destroys speech, and to localize speech are two different things. The damage is in my experience always in the region of the corpus striatum." This statement of Jackson as pointed out by Head is strangely in sympathy with Marie's localization of the lesion in aphasia in the quadrilateral area bounded by that author. Another worker on this subject, James Collier[5], expresses his dissatisfaction with the handling of this subject in the following words:

"The many schemes with their accompanying diagrams of the localization and interconnections of the speech-centers, that have been brought forward by high authorities upon this subject, have been founded more upon theory than upon trustworthy pathological evidence; upon these schemes classifications of the clinical varieties of aphasias have been based, which from the first have been open to the criticism that varieties of aphasia were included and described which were purely hypothetical and which had not been met with clinically. These were types of aphasia which should have resulted from lesions in certain situations, had the scheme which gave them birth been a correct representation of the physiological mechanism of speech. The multiplicity and complicity of the schemes and diagrams that have been brought forward are in themselves a proof of the difficulties that have arisen in the attempt to apply them to clinical cases."

Clinical studies of this subject, together with correlated pathological research, have failed to clear up the many problems involved. The area for motor speech in the third left frontal convolution, the auditory sensory speech area of Wernicke in the temporal lobe, and the visual psychic area in the angular gyrus, together with areas added later for writing in the second left frontal and an area embracing both frontal lobes as the seat of the concept center, all of them have been upheld by various writers reporting clinical and pathological material to sustain the individual views of the presenter.

Nevertheless, the concepts were so unconvincing, and the clinical and pathological material failed so signally to clear the situation, that Pierre Marie[22] demanded a revision of the whole subject of aphasia. He desired to sweep away the majority of preëxisting ideas upon the subject, and he railed against the conceptions advanced that speech memories are registered in the cortex as visual word memories, auditory word memories and motor word memories. He denied the existence of any of these centers, including the visual speech center in the angular gyrus or the supramarginal convolution, the Wernicke center in the posterior two-thirds of the left second temporal convolution, and he denied absolutely that the left third frontal gyrus had any function whatsoever in connection with the speech mechanism. He recognized no distinction between motor and sensory aphasias, and he argued that there was only one speech center diffusely localized in the left temporoparietal lobe and that this center is a region of intelligence specialized for language and not solely a center of sensory images. He insisted that the so-called word blindness and word deafness are defects of a special intelligence for speech. He recognized only one variety of aphasia from a lesion of the cortex and this is the type which is usually called the sensory aphasia of Wernicke. This type, he stated, always resulted from a destruction of the region of specialized intelligence for language situated in what he called Wernicke's zone.

The only other types of aphasia which existed for Marie were anarthria and pure alexia, which were always due, according to him, to subcortical lesions. The motor aphasia of Broca he regarded as a combination of aphasia with anarthria, due to a double lesion, one in the special speech intelligence area above mentioned and the other occurring within a quadrilateral area surrounding the lenticular nucleus, which area determined the anarthria. This quadrilateral area is limited anteriorly and posteriorly by vertical frontal planes which are level with the anterior and posterior limiting sulci of the Island of Reil. The outer limit is the surface of the insula and the inner limit is the wall of the lateral ventricle. Above, this quadrilateral area was prolonged to include the overlying gyri of the convexity of the cortex, while below it included the subthalamic region. The motor aphasia of Broca which he designated anarthria is usually due to a lesion in the upper two-thirds of this quadrilateral area which is in connection with the third frontal convolution, but contains no part of it and is in connection with Wernicke's zone also. That Marie certainly had sufficient reason to call for a revision of the whole situation as it touched our conceptions of aphasia, would appear from the highly important review by his associate Moutier[23], whose statistics are worth quoting:

"Between the years of 1861 and 1906, 304 cases with autopsy have been published upon the subject of the third left frontal convolution, or Broca's motor speech area. Of these, 201 cases were not of value for statistical purposes because in 26 of these cases the clinical or pathological records were incomplete and in the remaining 175 cases the lesions were too vast for accurate localization. There remained, therefore, 108 cases in which the lesions were localized, the records and clinical histories being of sufficient detail for use in the study of the subject. Of these 108 cases only 19 showed facts which appear to support Broca's theory, and in 11 of these, the lesion was not cortical at all, but was found in the subcortex. Of the 108 cases which were considered of sufficient value, only 8 showed the cortex of the left third frontal convolution to be affected. All the other cases were almost entirely against the classical theory that Broca's area was an area for the motor mechanism of speech. Including the 11 subcortical cases, only 19 were apparently favorable to this theory and even in a number of these cases there were ample lesions. In one there were extensive lesions of the right temporal lobe with no mention as to whether this patient was left-handed or not."

Both Marie and Moutier agree that there does not exist in medical literature one observation of Broca's aphasia in which a single lesion strictly localized to the foot of the left third frontal gyrus has been proved to exist upon autopsy. Indeed, workers in this country, Frankel and Onuf, analyzed 104 cases of aphasia with autopsy and met with only 4 cases which seemed to confirm Broca's localization; but at the same time they examined 5 cases of Broca's aphasia (motor aphasia) in which the left third frontal convolution was absolutely intact and the fibers going to and coming from this part of the cortex showed no trace of degeneration. A surgeon, Burckhardts[2], operated on a patient removing 5 grams of the gray matter from the foot of the first and second left temporal gyri and no word deafness (sensory aphasia) resulted. Eight months later, in the same patient, he resected the top and foot of the left third frontal convolution and no aphasia or agraphia resulted. In another case, the same surgeon removed a part of the left supramarginal convolution and destroyed a considerable portion of Wernicke's zone and no aphasia or any sort of speech defect resulted. In this last patient, the left third frontal convolution was also resected.

The operations in both these cases were performed with the deliberate intention of producing auditory and motor aphasia to counteract auditory hallucinations and over-activity of speech in two insane patients. In this connection, during the summer, I had an opportunity to examine a patient operated on by Guadiani at the Italian Hospital for a supposed abscess of the brain. The left temporal lobe was bored and punctured in several places. In the course of the convalescence some cortical tissue necrosed and three pieces, one as large as a five-cent piece, sloughed away at subsequent dressings. For a few days following the removal of the largest slough the patient was somewhat paraphasic and made mistakes, occasionally choosing the wrong word during conversation. He had no alexia, no agraphia, spoke fluently and spontaneously and from dictation, and could recognize, number and name objects and write their use without difficulty. The only trouble from which he suffered was that occasionally in the course of a conversation he would misuse a word or would stop for several seconds attempting to find the right word to express his meaning properly. The paraphasia in this individual lasted about three weeks. Just before he left the hospital I saw him again and could find no defect whatsoever. Despite all this, there is considerable feeling, even amongst the recognized authorities, that Wernicke's area and Broca's area and the area for psychic vision are intimately involved in the mechanism of speech production, as the following quotation from Dejerine[6] will show:

"Dejerine, Ladame et von Monakow, Liepmann et Quensel, Mahaim, Dejerine et Thomas—ont montré que cette circonvolution faisait bien partie du centre cortical du langage articulé comme cela était admis jusqu'alors, et la même démonstration a encore été faite sur le vivant par la chirurgie cranio-cérébrale. Ce que l'on discute toujours et ce que l'on discutera encore longtemps, c'est de savoir si le centre du langage articulé est étroitement limité á la circonvolution de Broca ou si, ce qui pour moi est plus que probable, il n'emiéte pas sur les régions voisines."

Enough has been shown even in the above quotation from Dejerine, to destroy any faith whatsoever in the conception which relegates the whole or any considerable part of that special skilled act, which we call speech, to definitely or diffusely circumscribed localizations in the cortex. It appears possible, however, that on the motor side the center for the innervation of the actual muscles concerned in the production of sounds for the service of speech, is situated in the third left frontal convolution near the areas for the larynx, pharynx, lips and mouth, and that they serve for the discharge of motor impulses cotemporaneously with the necessity for the expression of thought, which is speech.

A clinical report is hereby given of injury to the left temperosphenoidal lobe with recovery of speech function after slight temporary loss which may have been due to fatigue, to shock following operation or to the diaschisis occurring according to the theory of von Monakow, but surely not to loss of cortex substance as the absolute recovery seems to show. The questions used in testing are the work of Dr. Tilney and are employed by him in the Vanderbilt Clinic and in private work. A copy follows. The first report is of the examination ten days after the injury caused by operation and the second about three months after the injury:

Question 1—Can the patient speak spontaneously in ordinary conversation? (What is your trouble?)

Question 2—Can he enumerate and denominate, number and name objects? (Can you count your fingers aloud? Can you name the members of your family or the days of the week? Can you name these objects?)

Question 3—Can he speak from dictation? (Can you repeat, "Now is the time for every good man to come to the aid of his party?")

Question 4—Can he read aloud?

Question 5—Can he read to himself and understand what he reads?

Question 6—Can he recite what he reads or what he has previously learned?

Question 7—Can he write spontaneously?

Question 8—Can he enumerate and denominate in writing? (Can you write the numbers from 1 to 10? Can you add up this column of figures? Can you write the days of the week? Can you write the names of these objects which are shown to you?)

Question 9—Can he write from dictation?

Question 10—Can he copy printed or written matter?

Question 11—Can he hear and understand what he hears?

Question 12—Or sees?

Question 13—Is he aware of such errors as he may make in speaking or writing?

Question 14—Has he a concept of his own speech?

Question 15—Does he misuse words? Is he ungrammatical out of proportion to his education? Does he perseverate in fixed phrases? Does he speak a jargon?

J. de B., male, 46 years of age, Italian. Married, has five children.

He had an ordinary grammar school education in Italy and while there went one year to high school. He spoke and read Italian well, and was a prolific reader of Italian newspapers and novels. Since 14 years of age he has read no literature or scientific works of any kind. His education, therefore, is that of an average Italian youth of the middle classes. He speaks and writes English poorly and has never been educated in American schools, although he gets along fairly well with English. The examination has been conducted entirely in Italian.

Personal History.—He is right-handed and all his family are right-handed. Three years ago he suffered a small chancre for which he had nothing but local treatment. He has received no constitutional treatment for this condition since.

On April 10, 1917, he had a severe ache in the left ear with a slight fever. A day or two later the drum was incised and the ear discharged continuously for two months. At the end of this time the discharge ceased for three weeks, and his earaches and all the ear symptoms disappeared entirely for this period. At the end of July, 1917, he had another attack of earache in the same ear, together with which he had a pain in the head about the left ear within a radius of two or three inches. The headache continued with increasing severity, and finally his whole head pained him keenly. By the first of September, he had daily fever, never more than 101. He vomited for the first time during the month of July and this vomiting was attributed to some medicine which was being given. Toward the end of July, he had internal strabismus with diplopia in the left eye. He had at no time, until just following the operation, any disturbance of speech whatsoever. He was brought to New York to see his physician, Doctor Gaudiani, who considered that he might have an abscess in the brain or a brain tumor.

I was called in consultation August 29, 1917, and the examination at this time showed a double choked disc with swelling to the extent of 4 diopters in the left eye and 3 diopters in the right, left internal strabismus, tremendously severe headaches with tenderness, localized only poorly over the left side of the head, particularly marked on percussion over the left parietal bone and the occiput on that side. There was diplopia but no limitation of vision. There was slight deviation of the tongue to the left, and slight weakness in the muscles of the left side of the face. Taste in the anterior two-thirds of the tongue was preserved and there was no disturbance of function in any of the other cranial nerves. Sensation in the 5th distribution was normal. The superficial and deep reflexes were all active and equal, although to muscle testing at the wrist, elbow, shoulder, hip, knee and ankle on the right side as compared with the left, there was a marked weakness. Later on, this weakness became more marked and there was distinct pyramidal tract involvement with diminished superficial reflexes on the right side and more active deep tendon reflexes on that side. The headaches continued. There was no vomiting, no ataxia, no adiadochokinesis, no tremor, no convulsions, nor irregular, involuntary movements. The sphincters were well controlled.

Mentally, the patient was clear and there was no apraxia or aphasia. Despite my diagnosis of possible meningitic luetic process at the base of the brain, an operation was performed because of the antecedent ear condition. At the operation, no abscess or tumor was found, although a considerable amount of fluid gushed out when the dura was incised. Several deep punctures in the temporosphenoidal lobes in each of the convolutions exposed were made, and at subsequent dressings of the wound, three sloughs, two small ones and a large one about the size of a quarter, came away from the middle third of the second and third left temporal convolutions. Immediately following the operation, the patient's speech was absolutely well preserved, but one week later, September 18, 1917, he became paraphasic and an examination of aphasic status at that time, according to our scheme of 15 questions, is detailed as follows:

1. "I have a pain in the head. It is much better since the operation. I am talking fool-

ishly. I use the wrong words although I know what I want to say. It is laughable."

2. Counts aloud correctly up to ten. Does simple and even fairly complicated arithmetical calculations. Numbers and names objects correctly although on three or four occasions he said, for instance, "bed" for chair, and "writing" for fountain pen, and used the word "doctor" for nurse (dottoressa), which in English means female doctor. This was said in answer to the question as to who the nurse was. At times, too, he said his oldest daughter's name for his wife's name.

3. Repeats any number of test phrases accurately which are read to him from newspapers or will repeat long paragraphs fairly correctly, excepting that he will say "guerra" (war) for "battaglia." At times he becomes confused and will cry, and as a result his utterances for a few seconds will be very paraphasic. Words are misused and interchanged and ungrammatically arranged, but he soon recovers and speaks again with only an occasional misuse of a word.

4. He reads aloud correctly and without defect.

5. He reads to himself and gives a fair account, in his own words, of what he has read, occasionally only transposing one word or two in several long sentences. For instance, the last syllable of the word "presidente" is used alone or before the first syllable so that he will say instead of presidente, "dentoprese," or as he did on one occasion, "presidere." This does not happen very often and at times he will carry on conversation for several minutes without making any mistakes in speech. He often forgets his wife's name. I was reminded at the time of the similarity of his speech disturbances to those which are seen in fatigue. It must be remembered that this patient had suffered a great deal of physical and mental stress for several months and was undoubtedly exhausted, not only physically but mentally.

6. He recites perfectly what he reads, or test phrases that have been taught him.

7. He writes well spontaneously, except that he might use the same word several times in succession, and his grammatical construction is poor. Nevertheless, there is no jargon and the sense of what he wishes to say is very evident. As his writing is entirely in Italian I am not giving a sample of it.

8. He writes the numbers from one to any denomination. Adds up columns of figures correctly, writes the days of the week correctly, except that Sunday is written "domani" (tomorrow) instead of "domenica" which is proper. He wrote without any trouble or mistakes the names and uses of the objects shown him.

9. He writes well from dictation.

10. He copies perfectly printed or written matter and understands it.

11. He hears and understands what he hears and one must remember, particularly in connection with this entire examination, that this patient is very deaf as a result of his organic middle ear trouble. One is not always absolutely sure that he has distinctly heard what has been said to him. This seems to be proved from the fact that he obeys much more promptly and correctly written commands and answers written questions more correctly than he does spoken commands or questions.

12. He sees perfectly and understands everything that he sees and recognizes the use of objects shown him.

13. He is aware of such errors as he makes in speaking (in writing he makes none or exceedingly few), and becomes greatly confused and mortified and extremely emotional when he does make mistakes. He seems to feel that fun is being poked at him and will cry copiously after a mistake.

14. The number of syllables and letters in any number of words are perfectly recognized, particularly if written. The patient realizes that he makes an occasional mistake in speech and hastens to explain that he does not hear at all well and is tired and confused and has a profound headache, although he says, too, that the headache is not so bad as it was before the operation.

15. He misuses words in a paraphasic manner, although only occasionally, and in spoken speech he is much more ungrammatical than he is in written speech. There is a tendency to repeat phrases in writing or speaking, but in neither is there a jargon.

His few difficulties in speech cleared up remarkably rapidly. I felt at the time of his examination, and feel even more strongly now, that a great deal of his paraphasic trouble was due to fatigue, pain, exhaustion and actual difficulty in hearing. The ensuing examination made December 15, 1917, seems to bear this out:

1. "I feel pretty well excepting for an occasional slight headache. I still see two objects. I am very much hard of hearing. I can hear better than I did before the operation, particularly in my right ear. My left ear is the worse, but I can hear a little bit in it, whereas before the operation, I could not hear at all. My dressings (he still wears a bandage over the site of the operation) are always wet with fluid which comes from my head."

2. Gives correctly the names of his wife and five children, numbers and names objects in the room, gives the days of the weeks, counts the fingers on both hands, aloud, absolutely correctly.

3. Answered perfectly.

4. Answered perfectly.

5. Answered perfectly.

6. Answered perfectly.

7. Done perfectly: "Nel giornale della parola del Dottore, ho trovato che parlano di fare qualche cosa di benificienza pel Hospedale Italiano." *Translation*—In the Italian Medical Journal I find that they speak of attempting to aid in some way the Italian Hospital. Correct.

8. Well done.

9. Done perfectly. "Io sono de Benedictis chi ha sofferto molto nella malattia dell'orrechio." *Translalion*—I am de Benedictis who has suffered so much from sickness in the ear.

10. Well done.

11. Hears and recognizes watch, obeys perfectly spoken commands and many complicated directions of various kinds.

12. Sees well, has no hemianopsia and no limitation of vision, but has suffered from diplopia due to paralysis of the external rectus of the left eye.

13. He makes no errors either in speaking or writing except as a result of his profound deafness.

14. Lichtheim's test (Presidente, 10 letters, 4 syllables), correct. Ospedale (4 syllables, 8 letters), correct. He offers the information voluntarily that he is so hard of hearing and is so sensitive about it that sometimes he will not wait to make sure of what he has heard, but he is extremely intelligent and quick to answer when he hears well.

15. He does not misuse words, except that once or twice during the conversation at this examination when the title of the periodical which was used in the test, "La Parola del Medico" was mentioned, he said: "La Parola del Dottora." Dottore means just exactly what Medico means, so that this may not be a paraphasic manifestation. Before today he had never heard of the periodical mentioned. He speaks fairly grammatically, as he always did, never having been a perfect user of Italian. He does not perseverate, although quite naturally his thoughts as expressed in speech and writing are apt to turn towards his own difficulties, particularly his hardness of hearing. He speaks quite fluently and lucidly and not in a jargon.

TO BE CONTINUED

Owing to its length, the foregoing article has been divided into two parts; the second part will appear in the January issue of the BULLETIN. For convenience, however, the Bibliography of the entire work follows here.

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THE EXIGENCIES OF PEACE AS THE BASIS OF A WAR NEUROSIS*

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In reporting this case I wish to bring out some interesting, but as yet not generally accepted, data concerned in the symptom formation of the disease from which the patient suffered. I wish specially to emphasize, and hope to demonstrate clearly, that the symptoms in this patient are not isolated phenomena brought into existence with the onset of the illness, but that they have a very close association with certain phases of the patient's emotional or affective life preceding the onset of his illness. I shall also attempt to show that the symptoms from which the patient suffered are, illogical as it may seem on the surface, in reality an immediate gain to him, though he was unaware of this at the time. Lastly, I shall show that symptoms are repressed with fulfillments of an unconscious nature, indicating an actual regression to early psychological, or, more correctly, affective states.[1]

S. L., 24 years of age; discharged from the army in March, 1918, on account of illness, came to the Vanderbilt Clinic toward the end of the same month, having suffered from "spells" since the middle of January, 1918. The spells varied in frequency from 12 to 18 a day. The first one, as did all the succeeding attacks, came on very suddenly while the patient was reading a book. The patient was at home on leave from camp at the time. Suddenly he dropped his book, rose and walked aimlessly around the room. When addressed by his brother-in-law, he stated that he was all right, sat down and resumed his reading. Soon, however, he felt "queer," started to his room, and was unconscious for some two hours, when he awoke in the ward of a hospital.

The patient described the attack as follows: A queer feeling in the region of his heart, a severe pumping and pulling "as if his insides were all knotted up"; the sensation passed down into the region of the epigastrium and then to his right side. At the same time he felt a "drawing" in his left eye, his sight became dim, his head was forcibly turned to the right. Then his right hand was "drawn" to the region of his heart, and he pounded the precordium with a sensation of relief. After this he knew nothing until he awoke in the hospital.

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[1] Whenever I use the word "unconscious" in this presentation, it is with the meaning given it by Freud; namely, that an unconscious thought process is one of which the patient is unaware at the time. This thought process has been repressed into the unconscious because its contents are of a kind which carry with them wishes, emotions or feelings which we have learned are of a nature not compatible with a proper moral or ethical standard.

* Presented before the Clinical Conference of the Neurological Department of the Vanderbilt Clinic, May 27, 1918.

The family history is negative for any psychosis or epileptiform attacks. The mother is still living and is well, though of a very emotional, superstitious nature. She has had two illegitimate children. The father was accidentally drowned in 1903. There are a number of brothers and sisters, all of whom are apparently well.

The personal history of the patient, in so far as it reflects his attitude toward his environment and the effect on him of environmental influences, throws a great deal of light on the nature of his illness. It is this conception of the nature of the neuroses which has enabled us to recognize the true psychogenic character of such disturbances.

I shall give in brief those incidents in the life of our patient which have had a determining influence on the occurrence and nature of his illness.

The patient is 24 years of age, was born in the United States, and is the favorite of eight children. He was a premature infant, and on that account, as late as his twelfth year, he was made to feel that he was weak and small, not as capable as other children. He was coddled, petted and shielded to a degree that left a strong impress on his character, bringing to the fore a dependency, and a deep-seated feeling that he must be very carefully and tenderly treated. His mother, whom he idolized and idealized, had a most remarkable effect on his development. She was a highly emotional woman of Irish descent, intensely superstitious, and a firm believer in the potency of a curse. The patient was similarly constituted.

The boy went to school until he was 14. He left before graduating, because for two or three years preceding this time he had begun to lose interest in his work. He was a good scholar up to the age of 12, but later he found it very difficult to concentrate his mind on any work except drawing. The two years between 12 and 14 were fraught with great importance to the boy. At 12, his father was drowned under tragic circumstances. In the presence of the patient he had beaten his wife who had just risen from a sick bed. Thereupon his wife had cursed him and expressed a hope that he would not return from his next voyage (he was cook on a boat). The next news the family had of the father was that his body had been found in the river. Soon thereafter, the patient's mother consorted, without marriage, with a stranger. Our patient saw the couple in the sexual act on several occasions. Though he was but little more than 14 years old at the time, and had brothers much older than himself, without consulting anybody he reported the proceedings to the Gerry Society of New York City, swearing to the accuracy of his statements. The family was disbanded. The three younger children were put into an orphan asylum and the patient went to work, inasmuch as he took it on himself to pay for the support of the little ones.

Never of a frank, joyous nature, he now became even more moody than before, and when through with his work, which for two years was that of a hired boy on a farm in Staten Island, he spent most of his leisure time brooding over the downfall of his mother and in building day phantasies of revenge on the man who, he felt, had wronged her. At 16 he went to work in a factory and made his home from that time up to September, 1917, with an aunt, a severe, cruel, bigoted woman, who kept the patient in complete subjugation. Secretly he nourished plans for revenge on her, and had frequent fancies of burning the house, killing his aunt in numerous ways and finally of leaving her; yet as a matter of fact he remained in the home of this aunt until he joined the army in September, 1917.

It was with mixed motives that the patient joined the service. Patriotism, the desire to do his share, was a very important one. Another, though not fully conscious to the patient, was a desire to leave his unsatisfactory home surroundings with his aunt and at the same time defy her; for she had threatened to do him bodily harm if he enlisted. Moreover, his aunt had sworn to the patient that in case he enlisted she would pray that misfortune should befall him.

In the summer of 1917 he became acquainted with a girl whom he married in December of the same year. The marriage was decided upon after many doubts, chief among which was his feeling of sexual impotence. This had been a matter of grave concern and secret shame to the patient. Though not actually impotent, he suffered from ejaculatio precox, and dreaded the discovery of this by his wife, picturing the humiliation he would suffer on the occasion of attempting the sexual act.

His marriage, in addition to the sexual factor mentioned before, was a source of anxiety and conflict from a religious viewpoint. Reared a Catholic, he was devout up to his fourteenth year, then became lax, and upon his marriage was converted to Protestantism. So deeply rooted, however, were the effects of his early training, that even at the time he was under treatment, he wore a scapular. His conversion was accompanied with no peace of mind, and later he felt that "no good will come of it." Moreover, when his aunt heard of his conversion, she predicted misfortune to the patient as a result of this act.

His army life gave evidence of the emotional state which was soon to manifest itself as a hysteria major. As on many previous occasions, he at first took his work with enthusiasm and met with the approval of his superiors. However, about the middle of October he found himself less able to do things than he had been before. He tired easily and could not concentrate on his duties. Bayonet drill had a queer effect on him from the very start. During these drills he at first experienced a great deal of pleasure and excitement, in which he actually had a mental picture of bayoneting a German soldier. Later these exercises were accompanied by a peculiar depressing sensation located in the epigastrium and perineum, and in the progress he had phantasies of himself being bayoneted by a German soldier.[2] About this same time, i.e., the middle of October, fears began to take form that the curses of his aunt would be realized. Once his nose bled profusely without apparent cause. At another time he hurt himself while tramping through the woods in camp. All this, and his inability to work, he attributed to the evil influence of his aunt. While walking at night, he thought at times that he saw his aunt behind him, and, frightened, ran to camp. These states were always of very short duration, never amounting to delusions or hallucinations, and were always corrected by reasoning.

A short summary of the patient's make-up will help to visualize the picture and will, I hope, enable us to see the relation between the life of the patient and the occurrence of the disease. We have a dependent, highly emotional, impressionable, superstitious boy, whose family life up to his fourteenth year was a tragic one. Thereafter he was under the influence of a harsh, cruel, domineering aunt, and could not summon enough courage to leave her home. To get out of her clutches, and to revenge himself on his aunt, as he put it, he joined the army, where, also, he had many difficulties to overcome, being only partially successful. His marriage was accompanied by a mental conflict that he could not solve. All this emotional struggle took place in an individual who, in his sexual life, showed a decided variation from the normal. A physical examination showed a negative result except for exaggerated deep reflexes. We are now in a position to point out the significance of the various elements in the picture.

The outbreak of the neurosis itself concerns us first. We say this was the yielding of the conflict going on in the patient. More important, it should be noted that the illness was, although not realized as such by the patient, a solution of his difficulties, for his neurosis effected in the first place his discharge from the army, which now had lost its attraction, but this was replaced by anxiety and inability to work. Secondly, as a sick man it was with full justification that he could not carry out his duties as a husband in so far as the sexual act was concerned. Now his impotence and lack of sexual desire were the result of an illness and not the evidence of sexual weakness, as he had felt them to be before his illness. There are other immediate gains achieved by the neurosis. I have mentioned these two as the most prominent.[3]

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[2] It is interesting to note in this connection that, when boxing with one of his comrades, the sight of blood gave him intense joy and a desire to get a taste of blood. This was accompanied by bodily sensations located in the region of his genitals. Another interesting fact is that in wrestling bouts with his mates, the contact of his body with that of his opponent brought on an erection of the penis. I mention these incidents because we have learned by experience that in those who develop psycho-neuroses or psychoses, some variation from the sexual norm is, as a rule, demonstrated before the onset of the illness.

[3] These were motives totally unconscious to the patient at the time. It was only as the treatment progressed that he realized their import.

The regression to the infantile which is a characteristic mechanism in a neurosis will become evident from the following symptoms of the patient. An interesting feature of the illness was the gait of the patient. He walked on his toes, very carefully, as if fearful of making any noise. He kept looking to one side, head poised at attention, as if on the alert both to see and hear the better. The patient associated this with his stealthy comings and goings to and from his aunt's home at night, intent on escaping detection; for the aunt had frequently commanded him not to go anywhere without her permission. Fearful of detection, he, in similar fashion, often stole in and out of the house, intent on making no noise. In the home of his parents, he slipped stealthily in and out of the house, to associate with boys with whom his parents had forbidden him to go. Again on many occasions, with the utmost caution, he crept on his toes to the door of his parents' room to find out what might be going on there. Following the death of his father, the latter action was a frequent occurrence, leading to the detection and denunciation of his mother and her lover, who later became the patient's stepfather.

A symptom present on his first examination, which manifested itself as a rule after a seizure, and which lasted from 2 to 4 hours, was a semi-dazed mental state, during which his attention could be readily obtained, but held with difficulty. In this condition, he mumbled words such as, "You'll get yours," "I'll kill him," "Go to hell," "I'll kill her," "Don't hit me, oh, I'm so frightened." His facial expression corresponded to the thought content. A snarl and an expression of rage accompanied the first four exclamations, which had reference to the innumerable day phantasies in which he had indulged when vowing vengeance on his stepfather and his aunt. The last expression quoted referred to fear of his aunt, who had often struck the patient. The same mental states are again given expression in other attacks. In one of them, witnessed by me, the patient went through a bayonet drill, exclaiming during the procedure (accompanying the exclamation with the appropriate movement), "I'll kill him," "I've got him." His face at this point expressed intense hate and a fierce joy, as he fairly hissed the words quoted. In answer to the question "Whom are you killing?" he answered—"A German—my stepfather—my aunt." During another attack witnessed by me, his facial expression suddenly changed from anger to fear, during which state he exclaimed, "There she comes." In answer to "Who is coming?" he said, "My aunt, she wants to hit me with a chair." These were associated with frequent day phantasies of like content and actual occurrences in relation with his aunt.

When I last saw the patient in September, 1918, he had had no attacks for four weeks. These attacks had begun to diminish in frequency very soon after the patient began to realize the significance of the symptoms as pointed out to him. In the course of the treatment he frequently made his own interpretations, with consequent benefit.

I realize the incompleteness of this report. In spite of that, however, I feel that this case report is instructive from the standpoints I have indicated. I wish to add further that in general, similar mechanisms enter into the formation of the various psychoneuroses, but that symptoms vary in their manifestations, owing to the fact that they represent individual reactions to individual experiences or emotional impulses. The necessity therefore of individual study and treatment of a patient at once becomes apparent.

DISCUSSION OF DR. STERN'S CASE OF HYSTERIA

Dr. Michael Osnato.—I am afraid that Dr. Stern has been misinformed regarding his patient's military status. All the men at Camp Upton were drafted men up to a few months ago, so this one must have made a misstatement in saying he voluntarily enlisted last year. It seems to me there may have been a possibility of the man's trying to get away from the army rather than from his aunt, in that the attacks began after a month of camp life. The chief thing that strikes one about these psychanalyses is the fact that the patient's statements are accepted without question and apparently no attempt is made to confirm them. Neurotics are prone to be liars, and if this man is a pathological liar, it should be brought out. Some of the things he says he endured from the aunt are hardly explicable on any other grounds than that they did not occur. There is too great a tendency to accept at face value the statements of many of these neurotics and psychotics, and as the statements they make are the basis of many analyses, which in turn are the origin of psychanalytic fundamental laws, we see the possibility of great error and misconception.

Dr. Charles A. McKendree.—This case, which has been splendidly presented, is very interesting to me. I believe thoroughly in psychanalysis of certain well chosen cases and I think this case represents one of them. There are two or three points in connection with the history that attracted my attention. The first is that a man should tolerate such abuse from an aunt when there was really no obstacle to his leaving such an environment. It seems to me that there must have been a certain weakness in his morale. The second point is that this man, in order to escape from his hostile environment, should enter the profession of a fighter. Then suddenly, on very short acquaintance, he marries, which further complicates the picture of his mental processes. Then it is noted that the character of his work changes in a remarkable degree. I think that a man so mentally disjointed as would be indicated by the conduct of this one will bear constant observation, for there is a strong possibility that there may be some incipient psychotic disturbance at work.

Dr. Louis S. Aronson.—It seems strange that an individual accustomed to such warfare at home should fear entering the army. There are three possibilities in diagnosis in this case, epilepsy, hysteria and malingering. I do not think that Dr. Stern has excluded the last. The doctor has spent considerable time with his patient and we must assume that his findings are correct; still a malingerer can give just such a history, and go through just such attacks as this patient did. Malingerers of epilepsy can throw themselves into fits and their contortions closely simulate the convulsions of *grand mal*. I know of one case where the malingerer bit his tongue in order to produce a bloody froth from his mouth. It may take weeks of careful watching in an institution to make the differential diagnosis between hysteria, hystero-epilepsy, true epilepsy and malingering. It would be very interesting to watch this patient's future and as we gather some facts, compare them with the doctor's careful study of the past.

Dr. Frank M. Hallock.—It struck me, as Dr. Stern presented his case, how difficult it is to give the true picture of a case like this, bringing all the points together, even if one takes an hour or more to do it, and give a presentation halfway intelligible to those who have not seen the patient. It is an extremely difficult thing to do from an analytic point of view. It might be easier to take single symptoms at a time and try to work them all up in a way that would be understood by the neurologists, but these things are difficult for anyone who is even halfway familiar with the mechanism to understand. I always feel at a disadvantage in trying to make anyone understand them and hardly ever try to do so in talking about them. The only way to get an inkling of the relationship of such symptoms overlying a neurosis is to be in contact with the patient at the time one gets the enlightenment. Then it is so striking that one cannot fail to be convinced, but the impression is hard to convey secondhand. It is difficult to convince many people that there is anything in dreams or in dream analysis; I might argue there was something in it but would have no hope of convincing anyone. But if I could get coöperation with such a skeptic and analyze one of his own dreams, he would be convinced. It is very difficult to take a case like this one of Dr. Stern's and present it so as to give the real picture of the case and the meaning of the symptoms, for they certainly have a definite meaning.

Dr. Adolph Stern.—Dr. Hallock has stated what I would have said, namely, that it is difficult to present a complete picture of the case by giving only the bare essentials. Of necessity, in a short presentation, I have omitted a good deal which, if inserted, would have amplified and clarified some of the points in the presentation. When one takes into consideration that the life of an individual is not made up of disassociated experiences or thought processes, it becomes plain how such a short, almost disjointed presentation of the patient's life history is apt to lead to a misconception.

As for Dr. Osnato's suggestion that psychanalysts take for granted unconfirmed statements made by patients, I would say that as a matter of fact the main statements of the patient have been confirmed by his relatives (brother and sister-in-law). However, I want also to state that we analysts, just as do all people who come into intimate contact with delinquents, with the needy and those who are in some sort of mental conflict, learn to judge by the facial expression to find out whether the spoken word and the facial expression tell the same story or not. We also seek verification for statements. However, it is evident that most of what a patient tells must be judged on its own merits without recourse to outside aid.

Now as to the question of this being a psychosis, I believe we have no grounds on which to base that diagnosis. The semi-dream state which the patient presented on his first examination was that which he usually presented (according to statements of his relatives) on coming out of one of his spells. This symptom might suggest a postepileptic state (postepileptic confusion), the only form of psychosis entering into question.

As to epilepsy. The symptoms are against that diagnosis. The prolonged prodromal stage, with the complex nature of the prodromata, also their detailed description; the nature of the symptoms during the attack; the absence of unconsciousness, convulsions and loss of sphincteric control; all these are against the diagnosis of epilepsy.

Malingering is not to be thought of. The symptoms struck the examiner as absolutely genuine. Further, the more deeply we study hysteria the less likely are we to find malingering. Not that we are fooled by the malingerer. As a matter of fact, if one approaches these cases with, so to speak, a hostile or suspicious attitude, he will find himself greatly handicapped at arriving at the proper diagnosis.

As to the marked defect in an individual who will endure such cruel treatment from anyone for so long a time, I would say that we are all familiar with the individual who consciously desires tortures inflicted on him. Our patient has a very strongly developed masochistic element in him and this explains his "resistive submission" to the cruel treatment. I say "resistive" submission because in his fancies he often left his aunt, and inflicted on her all sorts of tortures, yet consciously submitted to everything and remained with his aunt. He joined the army to escape from all this, and married while in the service. Dr. McKendree stated that an individual who submitted to so many things would not be likely to join the army or to marry. However, I would call to his attention the fact that the patient did not succeed well in the army and also that one cannot say that so far he has made a success of his marriage.

I wish to state in conclusion that I hope to be able to analyze this patient, get at the cause of his abnormal emotional reaction to his environment, and effect a good result.

THE CAPSULO-THALAMIC SYNDROME WITH SPECIAL REFERENCE TO THE SENSORY DISTURBANCES

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and

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The case here presented offered an opportunity for more careful sensory tests than is ordinarily possible and is, therefore, of interest. The temperature tests are of particular interest in that they show a dissociation of extremes of heat and cold—a phenomenon that is said by Head and Holmes[1] not to occur in lesions of the thalamus—and also that the tests for cutaneous pain failed to show intensification on the affected side.

CASE NO. 16012.—Patient: D. L.; American; male; white; age 17 years.

HISTORY.—*Present Illness.*—Patient came to the Vanderbilt Clinic in October, 1917, complaining of continuous pain in entire right side of body, constant headache, and stiffness of right arm and leg.

The first symptoms were observed when the patient came home from a deaf and dumb school at Christmas in 1915, complaining of weakness in his right hand, but with no pain. While home, he was suddenly attacked by severe headache, lasting two days. The patient returned to school and finished the year. The school authorities reported that they were observing him and recommended an operation for brain tumor. On June 15, 1916, after he had returned from school, he was struck on the right side of the head by a horse, knocked down, and became unconscious. He felt the shock for several days. His hand gradually grew weaker during the summer, and he went for treatment to the Neurological Institute, Bellevue, Rockefeller Institute, and the Presbyterian Hospital; later he was sent to the Vanderbilt Clinic. He received treatment at the clinic for over a year during the latter part of which his condition progressed rapidly. Dizzy spells and a feeling of faintness were frequent, the right side felt constantly cold, he became despondent and had attacks of excitement during which he actually threatened his mother with violence. He was sent to the Presbyterian Hospital again in February, 1918, where he stayed for three weeks and was temporarily improved. His condition became complicated about this time by lobar pneumonia, with which he developed an empyema. He died at the Presbyterian Hospital, April 13, 1918.

Previous History.—Patient's mother stated that during pregnancy she was frequently beaten by her drunken husband; and that labor, while prolonged, resulted in a normal birth. Patient cried incessantly for three months, and, when three months old, was thrown downstairs by his father—to which occurrence his mother ascribes his deaf-mutism. The mother was unable to recall the facts of his development. He had colic frequently in infancy; diphtheria at three years, and pneumonia at four years of age; measles three times and whooping-cough twice while still very small.

He sleeps badly, is generally constipated, does not take alcohol nor use tobacco, and drinks coffee but once a day. He himself thinks his trouble resulted from his habit of masturbation. He spent about ten years in a deaf and dumb school, and appears to be fairly intelligent.

Family History.—The patient's father was an alcoholic addict. Patient had one sister who died in infancy from diphtheria. Otherwise family history is negative.

Examination at time of admission to the clinic in October, 1917, showed the following:

VOLUNTARY MOTOR SYSTEM.—*Attitudes.*—Position of right hand and forearm unusual.

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[1] Quoted by Jeliffe and White in *Diseases of the Nervous System.*

Gait.—Gait is spastic—right more than left. In walking with eyes closed, or in walking in a circle, he falls to the right. Fournier's tests normally performed.

Coördination.—Poor on right side due to spasticity. Pointing and pass pointing well done. No dysmetria. Adiadochokinesis present on right.

Skilled acts.—Marked dyspraxia in right hand.

Reflexes.—All reflexes, both deep and superficial, are active—in general right more than left. There is a slight Babinski on left.

Abnormal involuntary movements.—Athetoid position of right hand, due to athetoid mobile spasm.

Muscle strength.—Normal—possibly greater on left than on right.

Muscle status.—Muscles of right arm and leg are hypertonic. There is contraction of the flexors of right upper extremity excepting the fingers.

Abnormal associated movements.—The trunk thigh sign of Babinski is present, though slight, on right.

NERVE STATUS.—There is hyperexcitability. Chvoskek's sign is present on left.

GENERAL SENSORY.—The examination showed, subjectively, dull, constant pain on right side; objectively, complete loss of tactile sensation on right side, astereognosis on right, slight hyperalgesia, a diminution of temperature sensation on right arm, body, and face, and a tendency to call hot "cold." A more detailed account of sensory tests is given below.

CRANIAL NERVES.—*Olfactory and Nose.*—Subjective loss of sensation on right, normal on left.

Optic and oculomotor apparatus.—There is a right homonymous hemianopsia—mobility normal, fundi normal, no nystagmus, no diplopia, pupillary reflexes normal, but pupils slightly irregular—R, 4mm., L, 4.5 mm.

Acoustic and ear.—Not examined.

Trigeminal.—Motor: normal. Sensory: tactile sensation is absent, pain and temperature diminished.

Facial.—Sensory: not examined. Motor: paresis to involuntary movements on left, none when voluntarily contracted.

Glossopharyngeus, Vagus, Pharynx, and Larynx.—First was not examined; vagus apparatus normal.

Spinal-accessory and Hypoglossus.—Normal.

MENTAL STATUS.—Patient appeared to be quite intelligent. He understood and cooperated very well in the tests.

CRANIAL MORPHOLOGY. — Supraorbital arches prominent.

SYSTEMIC STATUS.—*Tegumentary.*—Hair is scanty on face, chest and axillæ; distribution of pubic hair is of female type. Skin shows slightly yellow pigmentation.

Glandular.—Small palpable nodes, axillæ and groin.

Cardio-vascular.—Blood pressure: 124, systolic; 74 diastolic.

Respiratory.—Normal.

Gastro-intestinal.—Normal.

LABORATORY TESTS.—*Wassermann.*—Negative.

Lumbar puncture.—Spinal fluid 23 cells. Otherwise negative.

Urine.—Negative.

Between January and March, 1918, a number of sensory tests were made, beginning with an investigation of temperature reaction by the double stimuli method.[2] Unfortunately, all the desired tests were not completed before the patient's final illness.

Deep pressure pain was examined by the Cattell algometer, increasing the pressure slowly and continuously until pain was reported. The patient was instructed to speak as soon as the pressure really hurt. Each area was tested three times, with intervals of five minutes or more between successive stimulations of the same spot. The variations between the different readings were, however, considerable.

Cutaneous pain and point stimulation for tactile sensation were tested by means of a sharpened bit of wood attached to a small spiral spring and connected with a scale showing the pressure in grams. The spring registered up to 25 grams. The method was as follows: Sixteen spots in an area half an inch square were marked by means of a stencil on the skin in ink. On the leg, eighteen spots were chosen, in two neighboring areas of nine spots each, each area one-quarter inch square. The wooden point was set down successively on each spot of an area so marked and pressure applied slowly (about one gram per second) until the patient reported that "touch" was felt. Pressure was continued until "prick" was reported, or until the limit of the spring was reached.

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[2] Described in NEUROLOGICAL BULLETIN, March, 1918.

The pain from a faradic current was tested with a Wappler machine by applying a current below the limen of sensation, and increasing the amount by moving the secondary coil one-half centimetre for each application until pain was reported.

Tactile sensitivity was tested both by point stimulation—as described above—and by areal stimulation, the usual clinical method of brushing the skin with a wisp of cotton wool being followed in this case.

Localization of a single point was tested by touching any spot on a region of the skin previously indicated (the patient's eyes being closed) with an inked point, after which the patient tried to touch the same spot with a similar inked point. The amount and direction of the errors were noted and the average for each region calculated. Unfortunately, the time at our disposal made the number of cases for each region too small to be very reliable. This test has the advantage of not requiring speech from the patient, but this is offset by a double disadvantage, in that localizations on the left arm had to be made by the extremely spastic right hand, while stimulations on the right side required a pressure sufficient to give the sensation of prick in order to be felt. It is probable, therefore, that the process of localization was not the same on the right side as on the left, and the test is of questionable significance in this case.

Temperature sensitivity was the most carefully tested. The reaction-time for temperatures of 17°–20°C. and of 40°–43°C.—or what Head calls extremes of heat and cold—was measured, and also the reaction to a double stimulus of these temperatures. Forty areas of the body were tested in a mixed order on four different days, and the median reaction-time values calculated for each region.

SUMMARY OF RESULTS OF SENSORY TESTS

Deep pressure pain.—On right upper extremity every area gives pain at a lower pressure than the corresponding area on left, the difference being more than a kilogram except in two cases, where it was 8 kg. The lower extremities showed a much less marked effect: on lateral surface of legs, *right* gave somewhat lower values; but on dorsal surface of foot, *left* was slightly lower. The differences were not large enough to be significant. The *range*, however, gave lower limiting values on both arms and legs, for the right side.

Cutaneous pain-prick.—This test shows clearly the diminution of sensation on the right side, but shows no intensification of sensation of prick. The average pressure required to give prick on the left hand was 7.2 gr. Every spot responded in both trials, and the *maximum* value was 13 gr. On the right hand, there were four spots that responded in neither trial with pressure up to 25 gr., and nine that responded only once; the average of those that responded was 22.6 gr., and the *lowest* value 15 gr.

On the lower extremities, the left gave an average of 11.5 gr., four spots failing to respond in one trial, and the range otherwise being 5 to 24 gr. The right showed eleven spots that failed to respond in two or more trials; and in only one instance, less than 25 gr. It was noted, however, that several times the response was delayed—it came as the pressure was removed. Possibly lower values might have been obtained with a slower rate of application or with a discrete series.

Electric sensitivity—pain from faradic current. On the dorsal aspect of the fingers, the current was felt later on the right hand than on the left; but all other parts tested gave lower values for the right side, and the difference was greater. The right side frequently showed delayed conduction, the interval between stimulus and response being sometimes as much as three seconds. The right side was consistently reported to hurt more than the left.

The pain limen, then, is lower on the right side for deep pressure and for faradic stimulation (except on the fingers), but higher for cutaneous pain. The two latter tend to show delayed conduction.

Light touch—point stimulation.—On the left hand, the average pressure required for touch to be sensed was 2.8 gr.—two spots failing in one trial to give any sensation of touch before the sensation of prick was elicited. On the right hand no touch sensation was reported. On the left leg, the average was 5.3 gr.—four spots failing to respond in one trial. On the right leg, no touch was reported.

Light touch—areal stimulation.—The lightest brushing with a wisp of cotton wool was felt normally on the left side and in the mid-line of the thorax and back of neck. It was not felt on the right hand, forearm, or arm (though here a slightly increased pressure was felt as tickle), nor on the right side of neck, nor on the right thigh, leg, nor foot, except along the tibia; but on the right cheek the lightest touch was felt as tickle.

Tactile sensitivity is, then, absent on the right side.

Localization of one point.—The average error in localization is greater on the right side than on the left for all areas except the right hand, but the difference is not greater than the mean variation and is therefore not of much significance. The greatest difference appears on the right forearm, where the average error is nearly three inches, although the variation is also great.

Analysis of the direction of the errors would seem to indicate a general tendency to localize touches on the left side *above* more often than *below;* while on the right side the two errors are about equal. Touches in the mid-line (forehead and sternum) tended to be localized to the left. The localizations on the right hand and arm showed an error in the radial direction, while those on the left showed an error in the ulnar direction—whether the arm was pronated or supinated.

Temperature sensitivity—double stimuli test.—The test shows a marked disturbance of hot on the right side. Hot stimuli gave only "cold" on the right side, except on the right cheek, where cold was perverted. These perverted cold responses were considerably slower in time than correct cold responses; but that they were not reactions to paradoxical cold arousable by hot stimuli, but true perversions of hot sensations, is shown by the fact that a temperature of 36°C. also felt "cool" on the right side although "rather hot" on the left. The cold stimulus showed no significant difference between right and left. In double stimulation, the perversion of hot prevented the perception of the double stimuli on the right side, although on the left it occurred with normal frequency. The time of reaction, however, was much slower than normal, owing in part, at least, to the profound speech defect of the patient. It is, however, noticeable that the responses that were given in the order *hot-cold* were—contrary to the normal type, and to other disturbed types—more prompt than those that were *cold-hot.*

There was some evidence of an increased effective reaction on the right side. On the right arm and leg, the stimulus was not infrequently followed by a reflex withdrawal of the part, which did not occur on the corresponding regions of the left side—though on abdomen and back, reflexes occurred on either side. These more often accompanied a cold stimulus than a hot one, but were as frequent with double as with single stimulation.

TABLE 1.

DEEP PRESSURE PAIN—(AVERAGE OF THREE TRIALS, IN KILOGRAMS, FOR EACH AREA)

Hand	L	R	Leg	L	R
dorsal-ulnar	3.3	1.5	lateral (1)	4.8	4.3
radial	2.7	1.5	(2)	3.2	2.5
palmer	4.5	3.7	Foot—dorsal	2.7	3.0
Forearm—volar (1)	3.5	2.5	Forehead	3.8	
(2)	4.3	2.0	Sternum		
dorsal (1)	5.8	2.5	anterior	3.8	
(2)	4.0	1.7	posterior	71.5	

TABLE 1 (*continued*)

DEEP PRESSURE PAIN

	L	R		L	R
Arm—dorsal	1.5	2.3	Neck—dorsal	4.0	
Upper extremity—av.	3.8	2.1	Lower extremity—av.	3.6	3.3
m.v.	1.16	7.9	m.v.	.87	1.02
range	1.0–8.0	.5–5.5	range	2.0–6.5	1.0–4.5

TABLE 2.

CUTANEOUS PAIN-PRICK—(PRESSURE IN GRAMS)

	Av.	m.v.	Range	No. of Trials	Failures	Spots tested	Spots failing once	Spots failing twice or more
Left hand	7.2	2.27	4–13	32	0	16	0	0
Right hand	22.6	2.21	15–25+	32	17	16	9	4
Left leg	11.5	3.22	5–25+	36	4	18	4	0
Right leg	25	0.45	15–25+	54	32	18	6	11
Sternum	10.5	5.56	2–25	32	7	16	7	0

TABLE 3.

PAIN FROM FARADIC CURRENT—(AVERAGE OF TWO OR THREE TRIALS, FOR EACH AREA, IN NUMBER OF CENTIMETRES MOVED BY SECONDARY COIL)

	L	R		L	R
Fingers— 2nd	1.8	2.0	Leg—lateral	3.1	2.6
(dorsal) 3rd	2.0	2.3	tibia	3.8	1.0
4th	2.1	2.7	Foot—dorsal	3.8	3.0
5th	2.0	2.6	Sternum		
Ulna protuberance	3.4	2.3	anterior	3.8	
Forearm—volar (1)	2.8	1.5	posterior	3.6	
(2)	1.9	1.3			

TABLE 4.

LIGHT TOUCH—POINT STIMULATION—(PRESSURE IN GRAMS)

	Av.	m.v.	Range	No. of Trials	Failures	Spots tested	Spots failing once	Spots failing twice
Left hand	2.8	1.17	1–5	32	2	16	2	0
Right hand	—	—	—	32	32	16	—	16
Left leg	5.3	1.78	1–12	36	4	18	4	0
Right leg	—	—	—	36	36	18	—	18
Sternum	4.2	2.72	.5–15	32	13	16	5	4

TABLE 5.

LOCALIZATION OF A SINGLE POINT—(AVERAGE ERROR IN INCHES)

	Av. Er.	m.v.	No. of Trials	Direction of Constant errors (no. of cases) Ce	Ca	L	R	M	La	Ra.	U.	V.	D.
Left hand—dorsal	1.36	.96	5	5	0					0	5		
Right hand—dorsal	1.25	.50	5	1	1					4	0		
Left forearm—volar	1.51	.38	10	1	6					2	8		
Right forearm—volar	2.67	1.20	10	0	1					8	1		
Left leg—lateral	1.36	.63	5	4	0							1	4
Right leg—lateral	1.73	.58	5	1	3							0	5
Left foot—dorsal	1.05	.34	5	5	0			1	2				
Right foot—dorsal	1.40	.38	5	3	1			0	5				
Forehead	.73	.28	5	5	0	5	0	3	2				
Left cheek	1.18	.53	5	1	2							4	0
Sternum	.52	.18	5	3	2	2	1	2	1				
Total on right side				5	6			0	5	12	1		5
Total on left side—				15	6			1	2	2	13	5	4

Ce, cephalad; Ca, caudad; L, left; R, right; M, mesial; La, lateral; Ra, radial; U, ulnar, V, ventral; D, dorsal.

TABLE 6.

MEDIAN REACTION-TIME, IN SECONDS, FOR SEPARATE COLD, SEPARATE HOT, AND DOUBLE STIMULATION

	Separate Cold Stimuli		Response 1st Intv.		Double Stimuli "Cold-hot" 2nd Intv.		Response "Cold"	
	Med.	P.E.	Med.	P.E.	Med.	P.E.	Med.	P.E.
Right arm	2.2	.25	—	—	—	—	2.2	.35
Left arm	2.0	.40	2.6	.55	1.1	.35	—	—
Face	2.3	.55	2.6	2.2–3.0*	1.0	.8–1.4	2.4	.00
Neck	2.4	2.3–2.6	2.9	2.2–3.6	1.3	1.0–1.6	—	—
Right leg	2.4	.45	—	—	—	—	2.6	.35
Left leg	2.6	.40	2.6	.65	1.1	.30	2.8	.15
Abdomen	2.3	.10	2.2	1.8–2.9	1.0	.9–1.2	2.6	.25
Back	2.5	.50	2.5	1.9–3.0	1.2	1.0–2.1	2.8	.70
	Separate Hot		Response		"Hot-cold"		Response "Hot"	
Right arm	—	—	—	—	—	—	—	—
Left arm	1.7	.10	2.0	.15	.8	.15	2.0	.35
Face	2.6	.25	2.4	.15	1.1	.50	2.9	2.9–3.0
Neck	2.0	2.0–2.0	—	—	—	—	2.1	1.6–2.6
Right leg	—	—	—	—	—	—	—	—
Left leg	2.9	.15	2.2	.30	.6	.10	—	—
Abdomen	2.7	—	2.0	.20	.6	.30	2.2	—
Back	1.7	—	3.2	3.0–3.5	.6	.6–.7	1.5	—

The following regions gave only response "Cold" to hot stimulus:

Right arm	Med.	2.8	P.E.	.40
Right leg	"	3.6	"	.95
Abdomen (right)	"	2.9	range	2.7–3.2
Back (right)		3.8	1 case	

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* Total range is given when there were less than four cases of a kind for any region.

CONGENITAL AMYOTONIA, PROGRESSIVE SPINAL MUSCULAR ATROPHY AND A CLASSIFICATION OF THE AMYOTROPHIAS*

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The subject of muscular atrophies as well as muscular dystrophies is at present in rather a confused state. Many authorities differ in their classification of the several clinical pictures and not infrequently similar diseases are grouped under different headings. Indeed, what appears to be one type of muscular atrophy is often divided into several distinct entities, with the embellishment of the name of the author who first reported his observation to add weight to the classification. Thus one speaks of Duchenne-Aran disease because the distribution happens to be in the hand, or Erb's disease because the shoulder is first involved, or Landouzy-Dejerine because the face and shoulder blade, etc., are attacked; or because the condition happens to begin at a certain age, the conclusion is drawn that a new entity has been discovered.

Without attempting to cavil or disparage useful classifications, it would seem that a firmer foundation can be had for the division of clinical pictures if it is sought in anatomy and pathology rather than in names of observers, chronological development, topographical distribution, or various ill-understood etiological factors, such as abiotrophy or even heredity and familial incidence. True, all these latter earmarks of the diseases are useful, and even necessary, for the identification of various conditions, but it does not follow that the underlying anatomico-pathological condition changes with the objective peripheral distribution. Therefore, we need not speak of different diseases when dealing with one background. A simpler classification, no doubt, would make for greater accuracy and better understanding and eliminate certain trimmings which, having originally served their purpose, are no longer necessary.

These few remarks are not to be interpreted as an attempt to discourage the recording of even the slightest variation from well-known clinical conditions. On the contrary, all observations and all variations, no matter how minute, should be carefully reported in order that many obscure points may be cleared up and our knowledge increased. It is unfortunate, of course, that so few of the cases under consideration offer their nervous systems to the microtome, but the clinical manifestations which are the more readily observed are by no means to be despised. The difficulty, however, lies not so much in failing to record anatomical substrata as in overestimating or lending undue weight to signs and symptoms.

Before attempting to discuss the subject under consideration and setting forth a few brief views, it may be well to report three cases. Two of them have something of interest, from the clinical point of view, to make them worthy of record, and the third one belongs to a sufficiently rare disease to render a description of it not altogether uninteresting.

PROGRESSIVE DEGENERATION OF ANTERIOR HORN CELLS WITH INVOLVEMENT OF PERONEAL GROUP OF MUSCLES

Case I; No. 21,128; P. G.; age thirty-five; male; white; married; native of Roumania; foreman in a silk shop.

Complaint.—Weakness in both legs and pain in back of the knees. The condition began in 1914, was gradual in onset, and has become progressively worse. At first the patient noticed that the left leg was weak and that the foot "flopped" on walking. The leg used to grow tired on standing for a long time. In 1916 the right leg began to grow tired and

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"flop" on walking. At present both legs are weak, and the patient has difficulty in going upstairs but none in going down. Of late he has complained of pain in the left calf and back of both knees. The pain is aching in character, but is not severe and does not trouble him at night.

The previous history is not eventful. The patient thinks he had convulsions in infancy and at the age of six is said to have had some paralysis of one leg, which lasted a few weeks and disappeared without leaving any weakness behind. He denies venereal diseases, and his wife never miscarried. His habits are good, he does not smoke, he takes no alcohol, he is normal sexually, he sleeps and eats well, his bowels are regular, he has no urinary disturbances, and vision is normal. He lost about thirteen pounds in the last year.

Family History.—Father died of endocarditis at fifty-six; mother is living. Maternal grandfather died of apoplexy, and a paternal uncle died of insanity, several sisters died in infancy. He has one child living and well.

Physical Examination.—Voluntary motor system. Abnormal attitudes and deformities; double drop foot, a left pes cavus and possibly also a right. The gait is steppage in character, more marked on the left side. There is no difference in walking with the eyes closed or open, but the patient cannot walk either on his toes or his heels. Coördination is normal in the upper and the lower extremities, both equilibratory and non-equilibratory, and there is no dysmetria or adiadochokinesis. Skilled acts are normal, and there is no disturbance of speech.

DEEP REFLEXES:

	R	L		R	L
Jaw	1	1	Suprapatellar	3	3
Pectoral	2	2	Patellar	3	3
Biceps	2	2	Achilles	0	0
Triceps	2	2			
Radial	2	2			
Ulnar	1	1	Periosteal (uncrossed)	1	1
Wrist	2	2	Periosteal (crossed)	0	0

SUPERFICIAL REFLEXES:

	R	L		R	L
Ciliospinal	1	1	Cremasteric	0	0
Supraumbilical	1	1	Plantarflexion	1	1
Suprapubic	½	½	Babinski	0	0
Upper lateral abdom.	1	1	Chaddock	0	0
Lower lateral abdom.	1	1	Oppenheim	0	0

Abnormal involuntary movements were absent. No fibrillations were observed. Muscle strength: marked, almost total loss of power in dorsal flexors of foot and toes on both sides. There is some diminution of power in plantar flexion of both feet and toes, L > R. The muscle status shows normal contour and soft consistency of all muscles of the body as of the legs. There is hypotonia of the muscles of both feet. The left thigh and leg are thinner than the right, one and one and one half c. m. respectively.

Electrical Reactions.—Loss of galvanism and faradism in the muscles of the left foot and peroneal group of the leg, and greatly diminished faradism and galvanism in the same muscles of the right. No abnormal associated movements. Nerve status, normal.

General Sensory.—Touch acuity, localization and discrimination, pain, temperature, vibratory, pressure, muscle-tendon and stereogenosis normal all over.

Cranial Nerves.—Olfactory, normal. Optic, oculomotor, trochlear and abducens; Vision $^{20}/_{20}$. Fields (roughly) normal, fundi normal. The pupils are normal in size and shape and are equal, react well to light and accommodation; no limitation of movements, no nystagmus and no other abnormalities. The acoustic, both cochlear and vestibular showed no abnormalities. The trigeminus, the facial, the glossopharyngeal, the vagus, the spinal accessory and the hypoglossus were normal.

MENTAL STATUS.—Normal.

Systemic:

Tegumentary, normal.

Glandular, normal.

Cardiovascular, normal; blood pressure 115 systolic, 76 diastolic.

Gastrointestinal, genitourinary and respiratory and skeletal, all normal.

Laboratory Tests.—Urine negative, blood Wassermann negative.

The diagnosis in this case is progressive spinal muscular atrophy. The interesting part is the peroneal type of distribution, which at first led to the belief that we were dealing with a so-called Charcot-Marie-Tooth type of neuritic muscular atrophy. The total absence of sensory changes, of course, immediately excluded the possibility of neuritic involvement and the want of other symptoms made the latter suggestion untenable. We are dealing, then, with a chronic, slowly progressive degeneration of the anterior horn cells; and it matters little whether we call this chronic anterior poliomyelitis or progressive spinal muscular atrophy. The point is that the muscular atrophy is secondary to degeneration of the secondary motor neuron; that is, we are dealing this time with a "nuclear" paralysis of the spinal cord. All other symptoms are secondary to that. The absence of fibrillations, the presence of a pes cavus and the history of some obscure leg paralysis in childhood furnish a little food for speculation, but the fact, nevertheless, remains that the case is one of anterior horn involvement, and apparently of that alone.

CHRONIC DEGENERATION OF ANTERIOR HORN CELLS WITH REMISSIONS AND IRREGULAR DISTRIBUTION

CASE II; No. 22,386; H. L.; age sixty-four; male; white; married; native of Germany; cashier by occupation.

On August 22, 1918, the patient came to the clinic complaining of headache, backache, and nervousness, saying that the condition began gradually about August 1, 1918. A little more detailed history, however, revealed a very different state of affairs. In 1894, he began to have weakness and tenderness in both hands, gradually extending to both arms. In the course of one year it progressed to complete loss of power if not absolute paralysis, so that his upper limbs hung limply by his side; he could not use them and had to give up work. The condition was diagnosed, so the patient says, by Dr. M. Allen Starr, as progressive muscular atrophy. After five or six months of treatment there was such great improvement that the patient was able to resume his work, though the better part of the atrophy persisted. For twenty years there was no change in the condition of his hands. In 1916, he began to suffer from headache and backache of an intermittent character, but in August, 1918, they became more persistent and he came to seek relief. He stated that in 1916 he noticed a weakness of his left foot and leg, gradually growing worse, but recently, synchronous with the exacerbation of his other complaints, his whole condition was very much aggravated. His whole history is not very clear as to the dates, but he is absolutely certain as to the time of the original onset in the hands, the remission of twenty years, and the gradual weakness in his left leg beginning some time in 1916.

Personal History.—He denies venereal and other diseases. In July, 1918, he had an iridectomy on the left eye, apparently for the removal of some growth (?). His habits are good, he does not use alcohol or tobacco, he sleeps rather poorly, but eats well, his bowels are regular, and he has no urinary disturbances. His vision in the right eye is normal. The family history is negative, his wife had one miscarriage (eighth pregnancy), and eight children, four of whom are living.

PHYSICAL STATUS.—Abnormal attitudes and deformities. Left drop foot, right wrist drop, hyperextension of left wrist. Gait, steppage left leg. Equilibratory coördination, normal; nonequilibratory also normal, except for some uncertainty in right finger to nose test. No dysmetria or adiadochokinesis. Skilled acts poorly performed R and L, due to weakness of both hands. Speech is normal. Abnormal in voluntary movements: slight, coarse, irregular tremor, possibly more on intention of both hands, R > L; no fibrillations.

DEEP REFLEXES:

	R	L		R	L
Jaw	1	1	Ulnar	?	0
Pectoral	1	1	Suprapatellar	?	?
Biceps	?	1	Patellar	1+	1+
Triceps	?	?	Achilles	1−	1+
Radial	0	?	Periosteal	0	0

Superficial Reflexes:

	R	L		R	L
Ciliospinal	1	0	Cremasteric	1	1
Supraumbilical	1	1	Plantarflexion	0	0
Suprapubic	1	1	Babinski	0	0
Upper lateral abdom.	1	1	Chaddock	0	0
Lower Lateral abdom.	1	1			

Muscle Strength.—Weakness on rising from recumbent position to standing, the attempt reminding one of the attitude in dystrophy. There is great weakness of the extensors of the right hand (wrist drop), weak flexors and extensors, abductors and adductors of both hands, weak abductors of right thumb and adductors of left thumb, weak extensor of left shoulder, very weak (almost 0) dorsal flexors of left foot.

Muscle Status.—Atrophy of extensors of the right hand and thenar eminence; atrophy of the ulnar flexors of the left hand, hypothenar eminence and interossei, also dorsal flexors of the left foot and calf muscles. There is atonia of all the atrophied muscles.

Electrical Reactions.—In the right hand, no faradic response in extensors and diminished response in flexors; no galvanic response in extensors, but present in the flexors. In the left hand, no galvanic or faradic response in the flexors and muscles of the hypothenar eminence, absent faradic and galvanic in peroneal extensors (dorsal flexors) of the left foot.

No abnormal associated movements. Nerve status, normal.

General Sensory.—Touch, pain, temperature, vibratory, pressure, muscle-tendon and stereognosis, normal all over. The patient states that he "feels better on the left side of the body," but this could not be made out objectively, so that it is rather doubtful. Cranial nerves are all normal. The left pupil shows an old iridectomy. There is pyorrhœa alveolaris. Mental status, normal.

Systemic.—Tegumentary and glandular, normal. Cardiovascular, pulse 78; blood pressure, 124 systolic, 78 diastolic. Respiratory, gastrointestinal, genitourinary and skeletal, normal. The blood Wassermann was negative.

The diagnosis in this case, too, is spinal muscular atrophy, and the interesting feature is the remission of 20 years. The distribution in the upper extremities is rather peculiar in that it seems to involve mainly 6 and 7C in the right hand, while in the left 8C and 1D (also a little 5C) are affected. The absent ciliospinal may or may not be due to involvement of 1D, as on account of the iridectomy it is hard to determine exactly.

As in the first case we are dealing with a muscular atrophy, probably due to destruction of anterior horn cells, and here, too, we need not quarrel over nomenclature. The muscle atrophy is secondary to chronic degeneration of the secondary motor neuron and to that alone.

Case III; G. S.; age six; female; white; U. S. The patient is a little girl who has not learned how to walk. She was born at full term, after prolonged labor and instrumental delivery. She was asphyxiated at birth but was soon resuscitated. The physician who delivered the mother states that the infant was born "all in a knot" and remained that way for some time. The limbs were flexed, and she lay in an intrauterine position, the muscles being very flabby. The general condition was poor, she had what looked like snuffles, could not nurse and had to be fed with a spoon. At six to eight months teething began; at one year she held up her head; she sat up at eighteen months, learned to stand when supported at three years, has never learned to walk, and began talking at eighteen months. She always was a clean child, has not been incontinent, developed mentally in a normal way, and is now considered bright for her age. Her speech has always been unclear, but she never had trouble in swallowing. She is irregularly constipated. The only disease she has had is measles. The whole condition has slowly improved.

The father is healthy but admits luetic infection. The mother is well. Her first pregnancy resulted in a still-birth, then she had two normal children, both living and well; the patient is the fourth, and the fifth pregnancy resulted in a miscarriage. It may be stated here that both the blood and the spinal fluid Wassermanns of the patient are negative. There is no parental consanguinity.

Voluntary Motor System.—Abnormal attitudes and deformities. The patient holds her head a little inclined to the left side. There is slight facial asymmetry. Gait and equilibratory coördination—the patient cannot stand. Finger to finger, finger to nose, and heel to knee tests show no ataxia. There is no dysmetria or adiadochokinesis. Skilled acts are carried out slowly, weakly but accurately. The speech is unclear, is not typical of any conditions involving either the peripheral speech apparatus or its central innervation, but reminds one of tongue-tie, although the frenum linguæ seems to be normal. There is no aphasia, no abnormal involuntary movements.

Deep Reflexes:

	R	L		R	L
Jaw	1	1	Suprapatellar	0	0
Pectoral	1	1	Patellar	1	1
Biceps	?	?	Achilles	?	?
Triceps	?	?	Dorso cuboidal	0	0
Radial	0	0	Periosteal (crossed)	0	0
Ulnar	0	0	Periosteal (uncrossed)	0	0
Wrist	0	0			

Superficial Reflexes:

	R	L		R	L
Ciliospinal	1	1	Plantar flexion	1	1
Supraumbilical	1	1	Babinski	0	0
Suprapubic	1	1	Chaddock	0	0
Upper lateral abdom.	1	1			
Lower lateral abdom.	1	1			

Muscle Strength.—She cannot rise from recumbent position to sitting or standing. She has difficulty in holding up her head, the neck muscles are weak, and a slight jar throws the head back. The muscles of the back are weak, and it rounds out in sitting. All the muscles of feet, legs, lower part of thigh, hands, forearm, and arm are weak. There is no paralysis or even paresis of any muscles.

Muscle Status.—Atrophy of the muscles of fingers, hands, forearms, arms, feet, legs and lower parts of thighs. The volume alone is lessened, but the contour is preserved. The atrophy seems to increase toward the periphery of the limb. The muscles are soft and hypotonic, show normal irritability and no myoidema. The electrical reactions show no qualitative alterations, but greater amount of current is generally necessary to induce contractions. All the trunk muscles are perfectly normal. Abnormal associated movements, absent. Nerve status, normal.

General Sensory.—Touch, pain, temperature, . vibratory, pressure, muscle-tendon, stereognosis, all normal.

Cranial Nerves.—Olfactory, normal. Optic, oculomotor, trochlear and abducens. Fields and fundi are normal. The pupils react promptly to l. and a. There is no nystagmus and no muscle palsy. The acoustic and trigeminus are both normal. Facial, normal, but there is a flattening of the right side of the face which is not due to involvement of the seventh. Glosso-pharyngeus and vagus are normal, but there is a deviation of the uvula to the right, without impairment of its movement. Spinal accessory is normal. Hypoglossus and tongue: she cannot protrude tongue very far out, the movements in all directions are normal, and the articulation shows interference with labials. The trophic status is normal. Mental status and cranial morphology are normal.

Systemic.—Tegumentary, normal; slight dermographia. Glandular, normal, thymus dullness not observed. Cardiovascular, respiratory, gastrointestinal and genitourinary, normal. Skeletal shows a very slight kyphosis.

Laboratory Tests.—Blood and spinal fluid Wassermanns, negative.

This case apparently is a typical amyotonia congenita of Oppenheim. The suggestion has been made that we may be dealing with an intrauterine poliomyelitis, but Oppenheim himself thinks that the condition is a pure myopathy. Certainly in this case there is no sign of involvement of the nervous system. Poliomyelitis may be excluded because there is no sign of segmental involvement of the cord, there is no atrophy of groups of muscles, the limbs are normal in contour, there is no R. D. and the early history is against the proposition. One does not see such symmetrical involvement in poliomyelitis. Although a luetic etiology might be entertained, as the father admits specific infection, it would seem merely a *post hoc ergo propter hoc* proposition in view of the negative serology in the patient.

SUMMARY

Although no pretense is made at a study of either progressive spinal muscular atrophies or the myopathies, the cases herein reported may be used as a text (or pretext) for a brief résumé and a short classification.

Without reference to etiology, chronic progressive muscular wasting may be divided into three groups. 1. Cases in which the muscular atrophy is primary. In these the salient feature is absence of any involvement of the nervous system. It does not matter what group of muscles is affected, whether it be in the limbs or trunk or head; it matters little what the order of involvement is, either chronologically or topographically. The point is that the muscles and they alone are objectively the seat of the trouble. All types are mere variations of the same conditions. It is idle, therefore, and confusing to set up a new entity, say, if the plantaris or pyramidalis nasi happen to be involved. 2. A very large group of obscure etiology in which the muscular atrophy is dependent upon the slow destruction of the lower motor neuron, be that in the cord, in the bulb or in the midbrain. The involvement of other systems is generally not considered, or we should have to include transition cases to a syringomyelia, amyotrophic lateral sclerosis, etc. The point is that there is a "nuclear" degeneration, as shown by atrophy, qualitative electrical change and no sensory or systemic disturbances. The common designation, therefore, of spinal, which is restrictive, should give way to nuclear muscular atrophy. 3. Cases in which the progressive muscular wasting is secondary to peripheral neural disease. The main feature here is involvement of the nerves. There may be slight changes in the cord, it is true, but they do not signalize the clinical picture. In this group are few well-defined entities, and it is doubtful whether it belongs altogether to the progressive muscular atrophies. But the point to be borne in mind is that beside the muscular atrophy there are sensory changes and that these changes are of a peripheral nature, that is not dissociative. The electrical changes are always qualitative and not primarily quantitative as in group 1.

With these fundamental distinctions one may proceed to classify the progressive muscular atrophies in any way he chooses. It were advisable, however, to throw out names of authors and substitute anatomical terms for them. Just as ventro-spino-cerebellar means more than Gower's, so does facio-scapulo-humeral mean more than Landouzy-Dejerine. However, in the subjoined classification the old names are retained.

GROUP I.

Primary Myopathies:

1. Primary Muscular Dystrophy (usually lower limbs).
   a. Pseudohypertrophic (Duchenne).
   b. Facio-scapulo-humeral (Landouzy-Dejerine).
   c. Bulbar type (Hoffmann).
   d. Juvenile scapular (Erb-Zimmerlin).
   e. Infantile (hereditary) (Leyden).
2. Myasthenia Gravis.
3. Amyotonia congenita (Oppenheim).
4. Myotonia congenita (Thomsen).
   a. Early life (Hereditary).
   b. Myotonia acquisita.
   c. Amyotrophic type (?).

GROUP II.

Progressive Nuclear Muscular Atrophy:

1. Involving small muscles of the hand (Aran-Duchenne type)
2. Hereditary familial type of early childhood (Werding-Hoffmann) } Spinal
3. Subacute and chronic poliomyelitis
4. Amyotrophic type (?)
5. Bulbar facial of childhood (Fazio-Londe).
6. Progressive bulbar palsy (Glosso-labio-laryngeal paralysis). (Bouchard).
7. Chronic progressive ophthalmoplegia (von Graefe).

GROUP III.

Progressive Neuromuscular Atrophy:

1. Progressive neuritic muscular (Charcot-Marie-Tooth).
2. Interstitial hypertrophic neuritis (Dejerine-Sottas).
3. Rare types of motor neuritis (?).

A BORDERLINE PSYCHOSIS WITH A DISCUSSION OF DIAGNOSIS AND TREATMENT*

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The patient, S. H.; thirty-five years of age; colored; married; came to the clinic on November 21, 1918, with the following history:

One morning in October, 1917, while working as a porter in the Pennsylvania Railroad station, he was struck on the head by a falling electric sign. He was rendered unconscious and remained so for about six hours. That same evening, after recovering consciousness, and two or three times for several succeeding days, he had an attack of unconsciousness preceded by a cry, with spasm of the right shoulder and arm. The convulsions then became general. There was loss of control of the urinary sphincter, with biting of the tongue. Sleep lasting about five minutes, followed this attack. Ever since the accident the patient has been practically mute, except when addressed, when his answers are usually irrelevant. He sits continually quite motionless, in a constrained, rigid, uncomfortable position. He has to be forced to eat. Headache, "dizziness in the head," are constant complaints.

A subsequent history, obtained on November 30, 1918, revealed that the urinary incontinence frequently was independent of the attacks and very often was followed by retention of the urine, as at times the patient had to be told to void his urine. The convulsions, on close questioning, proved to be nothing more than shrugging of the shoulders. The trembling of all the limbs was present even when the patient was not unconscious. The attacks of unconsciousness with their attendant phenomena, were preceded by a cry and came on suddenly while the patient was walking or sitting in a chair. He then fell to the floor and resisted all attempts to lift him. He remained in this condition about five minutes, and afterwards often fell asleep. In reality the biting of the tongue consisted of chewing movements, at which times the lips were sucked into the mouth. These actions were present on occasions other than during the periods of unconsciousness. Infrequently the patient gave some evidence of hallucinations by calling out "Go away from here," and "They are making a noise." At such times there were no outward causes for such remarks.

The previous history of the patient is that he was of normal intelligence, graduating from public school at fifteen. He was of a sociable, well disposed, cheerful make-up, not given to very marked emotional changes. There was no evidence of any neurotic or psychotic disturbances. He married in 1910, and has three normal children. The family history is likewise negative, as far as could be ascertained.

A physical examination showed the following:

VOLUNTARY MOTOR SYSTEM:

1. *Abnormal attitudes and deformities.*—The patient sits in a constrained, slouching, absolutely motionless position, with eyes half shut and looking steadily in one direction, head held to one side.

2. *Gait.*—The patient walks with trunk rigid, legs far apart, like an automaton.

3. *Coördination.*—(a) Equilibratory, standing with feet together, eyes open. With the attempt to approximate his feet, on different occasions, the patient falls to the floor, now in one, now in another direction. No coöperation could be obtained to elicit further evidence of a state of coördination or in regard to skilled acts or abnormal involuntary movements.

6. *Reflexes.*—No abnormality was evident. The abdominals could not be elicited.

8. *Muscle status.*—Negatavistic resistance to passive motion was obtained at times in all limbs which then remained on occasion in a cataleptic condition for a variable length of time.

D. GENERAL SENSORY.—The only tests carried out were pin pricks, superficial and deep, and deep pressure to muscles of limbs.

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* Presented before the Clinical Conference of the Neurological Department of the Vanderbilt Clinic, December 2, 1918.

The patient evidenced no reaction whatsoever to them, either by change in facial expression or attempt to draw away the parts so tested. He did not coöperate verbally.

E. Cranial Nerves. 2. *Optic.*—Pupils are equal, circular, central, react promptly to light. No other tests were carried out as patient did not coöperate.

5. In testing facial nerve and face, no evidence of disturbance was found. Patient, however, carried out movements contrary to those requested of him.

8. *Hypoglossus Nerve and Tongue.*—Patient very slowly protruded tongue when told to raise it.

F. Mental Status.—The patient does not look very intelligent, but it cannot be ascertained whether or not any deterioration has taken place. He displays no emotional play of features or expression whatsoever. His emotional tone is apparently that of complete indifference rather than of depression. The patient, in reply to questions as to how he feels, merely states that he has headaches. His attention is obtained with great difficulty and at once is lost. The expression of the eyes, however, when it can be seen, is that of furtive alertness. Memory tests elicited very vague replies or none at all. In answer to, "Where do you live?" he said, "Uptown." He gave correct answers as to age and length of time married. No replies to other questions to test his memory were obtained. The impression obtained by the examiner was that there is no memory defect present, and that the vague answers are evidence of his negativistic attitude. Nothing of a delusional or hallucinatory nature was obtained by questioning, nor did the patient, while under observation, show any facial or other reaction to hallucinations or delusions.

H. Systemic. 1. *Tegumentary.*—Skin was dry.

3. *Cardiovascular.*—Pulse slow, small, soft; heart sounds faint; otherwise normal.

6. *Genito-urinary.*—Bladder distended.

To summarize: In a previously normal individual we have the sudden onset of a condition following immediately upon a trauma, manifesting (1) epileptoid phenomena; (2) mutism; (3) constrained, uncomfortable attitudes, both (2) and (3) being maintained over long periods; (4) retention of urine, with occasional incontinence; (5) refusal to eat spontaneously; (6) headaches; (7) vague hallucinations of sight and hearing; (8) the persistence of these symptoms from the time of onset to date, without any material change.

The physical examination reveals (1) a constrained, uncomfortable, slouchy, absolutely motionless position when sitting, maintained indefinitely; (2) lack of coöperation in carrying out tests; (3) negativistic manifestations both in the physical and the psychical fields, with a mild cataleptic state at times; (4) no response to sensory stimuli of a painful nature; (5) mutism; (6) absence of all emotional display with apparent indifference; (7) no evidence on examination of delusions or hallucinations. Apparently the memory is good so far as it may be determined.

The question of diagnosis between a traumatic hysteria and dementia precox is raised, not so much by the clinical picture as we see it now, which certainly is strongly in favor of dementia precox, as it is by the mode of onset. The sudden onset, in a previously normal individual, following immediately upon a trauma, is very suggestive of a traumatic hysteria. Moreover, the continuance of the symptoms with the same degree of intensity and variety is also in favor of traumatic hysteria.

Another factor which I have not as yet mentioned is that the patient's case is in court to be tried very soon. The effect on the symptoms of the outcome of the case will aid to some extent in the diagnosis. If the patient becomes well soon after the suit is decided, the diagnosis of traumatic hysteria will be established. However, the continuation of the symptoms in spite of a favorable verdict, will not of itself necessarily rule out the presence of traumatic hysteria.

As I mentioned above the clinical picture as we now see it (the mutism, the negativism, the rigid, motionless, constrained attitude, the evidence of cataleptic phenomena) is that of a case of dementia precox.

DISCUSSION OF DR. STERN'S CASE OF DEMENTIA PRECOX

Dr. Charles A. McKendree.—This case is most interesting to me because it involves certain points in differential diagnosis to which I have given considerable attention. The first thing that suggested itself to me on seeing this case was this: Had the man been paid for his injury? If he had not, was the case in court? I think that is the most essential question to ask in all of these pictures. On the other hand, as the history was read, I was impressed by the fact that the man is really suffering from an organic injury. He has developed epilepsy upon his trauma, the symptoms of which he shows during seizures by biting the tongue, somnolence afterward, with true convulsive movements, and of which seizures recur at various intervals. I am sure we are dealing here with true epilepsy which has followed trauma. As to its being a dementia precox, we cannot get away from the fact that this man was entirely well until he was hurt, and that upon receiving his injury he developed this attitude of mind and body. Dementia precox does not assume the same phase constantly over such a long period of time. His attitude is one of chronic discontent against those to blame for his injury, and when his lawsuit is settled and he has received proper treatment, I believe he will assume a different mental and physical attitude. His mutism and negativism are possibly simulated, and anesthesia to pinprick all over the body, while not a common symptom of precox, is quite common in hysteria. It is very easy for a dementia precox individual to be hysterical and it is easy for an epileptic to be hysterical. Often we are quite satisfied with ourselves, when we find a patient has convulsive seizures, is not incontinent, knows what is going on, does not bite the tongue and has no headache, in regarding the case as hysteria. But those purely functional attacks certainly do occur frequently in true epilepsy. In the advisory board work we see many true hysterical outbreaks in individuals whom we know to be epileptics. While it is perfectly possible for the precox type of man to develop hysterical manifestations, nevertheless I consider this case a functional one with an organic epilepsy.

Dr. Frederick Tilney.—I would like to call attention to a case I presented to the fourth year class several weeks ago; a boy who was injured in a football game and subsequently developed epilepsy. He was brought in from a sanitarium to the Vanderbilt Clinic. The progress in the mental symptoms was so marked that before the day was over, it was necessary to call an officer and have him taken to Bellevue. The boy had manifested a marked psychosis, but there was a question as to the exact form he had. The particular point was this: Should we make a diagnosis of epileptosis or was this a schizophrenia. I watched him for an hour and concluded that he was the catatonic form of dementia precox. It is possible that an epileptosis may become a schizophrenia. I believe, in the case of Dr. Stern, we are dealing with something more profound than hysteria.

There is another factor to be considered in these cases. It will pay us to give attention to the vast changes which occur outside of the psychic domain. There is marked disturbance of the gastrointestinal tract; the autonomic and sympathetic system are involved parallel to the psychic factor. We overlook these too much.

Dr. Walter A. Bastedo.—If anything is found in this case that suggests toxemia, the questions of ingestion, elimination and destruction in the body are the three we must consider. If, for example, this man has intestinal putrefaction, as decided by stool examination, that may be contributory to the symptoms he manifests. Treatment of this condition might tend to overcome his absolute indifference, because in toxic individuals we frequently do see very marked indifference. In experimental animals of social type, it has been found that when injections of phenol, indol, etc., were given, there resulted a decrease in sociability and that as the doses were increased the sociability disappeared completely so that the animal remained by itself in another part of the cage, moving away when another approached. It may be that human beings are made unsociable or indifferent, to a certain degree, through similar intestinal poisons. Many years ago I began to give attention to these poisons and I have lived to see intestinal putrefaction, previously scoffed at, accepted as a serious matter. I have heard Coleman and others give it a place in the etiology of some of the bad symptoms in typhoid fever, and I have heard Lewellys F. Barker of Baltimore say that intestinal putrefaction was not only serious in typhoid fever, but might be the actual cause of nephritis. I, myself, am sure that it is a very frequent cause of mental depression.

The treatment consists largely of (1) elimination, by giving a great deal of water to keep the kidneys active and to favor the carrying out of toxins; (2) proper emptying of the bowels, lavaging the colon if necessary, by irrigations; and (3) taking from the diet for a sufficient length of time those foods that seem to be the sources of the material from which poisons are made. These, as a rule, are some forms of animal flesh and eggs, but there are other foods, such as peas, beans, cabbage and spinach, which in some cases furnish poisons.

In the matter of colon irrigations, ninety-nine out of a hundred are not properly given. There is only one way to tell, and that is to look at the results the nurse gets. As a rule, in hospitals the process is gone through with in about 20 minutes and the result is a stool such as one gets from an enema, a few flakes of mucus and mostly fairly clear fluid. It is thus apparent that the water has never been above the descending colon. Colon irrigations must go further up and usually should take an hour. Experimentally, I find that if water is run by a double return tube into the rectum with the patient lying on his back, the water will at first bring the ordinary contents of the lower part of the bowel; that after a time, perhaps 20 minutes, it will begin to bring the contents of the transverse colon; and that not for perhaps 20 minutes more will it bring the putrefactive material which one is after. Rehfuss obtained similar results. It seems impossible to get the cecum cleaned out in less than 40, 50 or 60 minutes. In most cases I prefer the double tube method to that of the single tube. It is only necessary to put the tube up three or four inches, for beyond this a soft rubber tube usually merely coils up in the rectal pouch. The frequency with which the colon irrigations are given is of importance. In my opinion they are used too frequently. I find it sufficient to employ an irrigation every second day for one or two weeks; after that once a week, or two days apart once a week will do all that can be accomplished to advantage with colon irrigation. It is much better to get the tone of the bowel right and to keep the intake of the poisons down.

Professor C. C. Lieb.—If it is true that putrefaction is most active in the lower part of the small intestine and the upper part of the large duct, in selecting a cathartic to be used in the treatment of intestinal toxemia the point of action of these drugs must be borne in mind. For instance, the anthracenes, such as cascara and aloes, will not do, as they act almost entirely on the lower part of the descending colon. Again, the salines are harsh, and rush through the intestinal tract too rapidly to produce a thorough cleansing. Calomel also is contraindicated; it acts chiefly on the small intestine, but is too toxic a drug to be administered day after day. Though the action of castor oil would seem to indicate its use in the treatment of intestinal toxemias, it is too disagreeable to take and is followed by constipation. The drastic cathartics, however, such as jalap and Podophyllum, are believed to act chiefly on the small intestine and the upper part of the large intestine. Therefore these drugs would seem to be especially adapted to the treatment of intestinal putrefaction.

Dr. Albert Polon.—Endocrinology might be thought of if this case had been established as dementia precox, for the extracts have been known to act in a specific way in these individuals; small doses of thyroid, for instance, seeming to bring about decided improvement. It has its influence on the movement of the bowels. To begin with the dose should not exceed one-tenth grain.

Dr. McKendree.—I should like to add to my discussion a thought that has just occurred to me. In the work of Irving J. Sands, some of which has been presented before these conferences, he reported several cases of psychoses, one of which I believe was a precox, in which neoplasm was found at autopsy. I believe that, following the real and serious trauma this man has suffered, one might reasonably think of neoplasm as the causative factor in this abnormal picture and therefore I think he should have a careful neurological examination including blood and spinal fluid and the eye grounds.

Dr. Adolph Stern.—This patient's lawsuit is coming up for trial next month, and it will be interesting to see if a definite result as to the outcome will have any influence on his mental state. In reply to Doctor McKendree, I may say that some of the symptoms this man presents are common to both precox and hysteria. There is no reason why we should not get symptoms of hysteria in a precox make-up, or vice versa. In spite of that, however, the symptoms of precox here seem so definite that the diagnosis seems clear to me. I have seen other precox cases present precisely just such emotionless attitudes, keeping it up for months at a time. There is no reason why this should not be a precox of the milder grade.

I dislike the use of the word simulation in connection with such patients as this. To my mind, there cannot be any simulation that will produce such a condition. I always conceive of a state such as this to be an involuntary, unconscious mechanism on the part of the patient, and I think we should discard the word simulation as rapidly as possible, when we will be in better position to understand the mental condition of the patient.

As to there being an epilepsy here, I do not think the history given me is one of epilepsy. I questioned the patient's friend closely, however, and his description of the attacks was not so suggestive of epilepsy as the first history taken. He now says that the patient falls off his chair and "jerks with his shoulders." This, too, may be incidental to his condition and not to epilepsy. I think the diagnosis of dementia precox is more likely than that of hysteria, although I am not so sure that we can rule the latter out.

As to treatment, I believe that which has so far been followed out can be continued. He has been taken care of as an invalid who cannot help himself. As he is at present, it is safe to keep him at home where I think he is better off than he would be in an institution. The treatment is symptomatic. I do not think that any psychotherapeutic measures would be of any avail, owing to the inaccessibility of the patient. His lawsuit should be settled as speedily as possible.

SOME VIEWS OF AN INTERNIST ON THE TREATMENT OF EPILEPSY AND HYSTERIA*

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The treatment of epilepsy and hysteria must be regarded from two points of view, viz., the general mental and physical condition of the patient, and the special manifestations which may come from time to time in the form of attacks. From the physical point of view there is much that can be done for a hysterical patient. For example, attention may be directed to the condition of the alimentary tract. Undoubtedly, in many cases, derangements of the stomach and of the bowels undermine the nervous stability. I have seen hysterical manifestations come on during such periods of digestive derangement and disappear when they had been properly treated. It is now an accepted fact that the poisons of putrefaction produced in the intestines have a great deal to do with undermining the nervous stability. That, alone, brings us to a large field in therapeutics, and I have not time to go into the details of its treatment here; but putrefaction is so often present in nervous diseases, particularly epilepsy and hysteria, that it would appear in these cases to be very important.

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Besides this, the general use of drug remedies for nervousness must be considered. When a hysterical condition stands out for periods of two or three days at a time, one uses such a drug as valerian. It seems to have an absolutely different effect from that of bromides, and probably its action is to increase the control by the higher cerebral centers rather than to depress it. In other words, it has a stimulating effect upon the cerebral processes. With a hysterical outburst the patient will sometimes calm down after taking a cup of tea or coffee, and it may be that valerian acts in somewhat the same way. It is generally believed that the drug gains this action at least in part through the sense of smell.

Among other measures that are undertaken frequently, leaving out forms of psychotherapy, we have hot and cold packs, quiet and freedom from annoyance, and occasionally a dose of bromide; all of which play a part in holding down these hysterical manifestations. Some persons have hysteria for a short period only and but once in a long while, though they are of a highly neurotic nature; others, no more neurotic, show quite frequently some form of the hysterical temperament. There must be a great difference in the form of therapy in these two groups. In those who show hysterics infrequently, we are called to treat nothing but the attack when it comes. But in the others we usually have opportunity to care for the underlying physical state. In these, my strongest point is to get after the alimentary tract.

Intestinal putrefaction depends upon whether there is hyperacidity of the gastric contents or anacidity. For permanent results, treatment of the latter condition is difficult. In some cases one encounters a persistent or recurrent type in which almost any protein seems to disagree, not only because the colon bacillus or other organism splits it, but also for other reasons; and one is unable for a long period of time to feed sufficient protein to keep the patient in a good muscular state.

Besides attention to the gastric condition, the cure of intestinal putrefaction implies several things, viz., 1, limiting the supply of material which yields putrefactive products; 2, emptying the colon; and 3, detoxifying the poisons, a function performed chiefly by the liver.

1. If there is bleeding going on we may have a putrefactive activity associated with the blood. But practically, the food is the source of the poisons. Therefore, the first thing to do is to cut off for a sufficient length of time those foods which readily putrefy. Such are the animal flesh foods and eggs, particularly raw eggs. Mendel and his co-workers have found that raw eggs do not digest well at all, even a lightly boiled egg being much more digestible. At times, too, other foods putrefy, for example, beans and peas, and this is particularly true when they have been dried, so that soups made from dried beans and peas, or baked beans, such as are so generally eaten in New England, are prone to produce putrefaction.

2. That the colon should be properly emptied does not mean that one should be content when the bowels move every day. It implies adequate movements without hastening or retardation. Cathartics which rush the contents of the stomach down into the colon too rapidly will favor intestinal putrefaction, because it is the raw protein which causes the trouble and the protein has not had time for digestion. On the other hand, if there is stagnation of the colon, there is opportunity for slow putrefactive changes to take place in its contents.

3. When these poisonous products are absorbed they must be rendered innocuous by the liver. In experiments made in my laboratory, Howland and Richards, under the guidance of Herter, showed that injections of indol and phenol into experimental animals always produced the same train of nervous symptoms, beginning with moroseness, and, if the dose were large enough, ending in convulsions. They then abolished the oxygenating power of the liver and found that the doses producing these symptoms needed to be much less than before the liver power was destroyed. It would seem that the liver function might be promoted by active hygienic measures. It was William Hunter who said that in order to avoid all forms of putrefactive poisoning we should have the intestines of the ox and the liver of the pig.

These three things, then, are important: Elimination of putrefactive proteins from the diet, an intestine which does not allow residue to remain any great length of time, and a good liver.

REVIEWS & ABSTRACTS

A Contribution to the Knowledge of the Pathological Neuroglia and its Relation to the Degeneration Processes in Nervous Tissue. By Alois Alzheimer. *See references at end of abstract.*

I. AIM OF THE INVESTIGATION

The author states his proposition that the ordinary staining methods—Nissl's[5] cell stain and Weigert's[9] glia and medullary sheath stains,—while they show changes in various disease processes, do not bring to light anything which can be called pathognomonic. The Nissl stain shows many different changes in the ganglion cells but not always the same changes in any single disease. Thus we know very little of the pathological value of the different Nissl pictures. It is difficult also to note the disappearance of nerve-cells as there are so many of them in the cortex that a few are not missed.

The same thing may be said of the nerve-fibers. Only massive fiber degeneration is noticeable, for when only a few are gone, the remaining fibers tend to crowd close together and thus obscure the result.

Glia stains show equally little that is characteristic. Weigert claimed that every distinction of nervous tissue was accompanied by an increase of glia and new formation of glia fibers. This is unfortunately not true, for much degenerated nervous tissue may contain fewer glia fibers than normal tissue.

Nissl has stated that definite nerve-cell changes are accompanied by definite changes in the glia. These changes do not consist of glia fiber formation but of progressive and regressive changes in the plasma and nucleus of the glia cells. Alzheimer,[1] however, considers that it takes the fine discerning eye of a Nissl to see any morphological connection between these changes.

Finally, the pathological changes in the mesodermal elements of the cortex are not so varied that differentiation between the psychoses is possible. The changes here do help much in the differential diagnosis of Paresis, Brain Syphilis, Senile Dementia and Arteriosclerosis, but similar changes occur in all processes when nerve-tissue is degenerating.

Again we often see patients with the severest cortical diseases or unrecoverable deteriorations which show very little at postmortem, and the changes that do occur are the same as those seen in the mildest psychic disturbances. Alzheimer considers all this due to the fact that our staining methods are not good enough to show changes in the finer structures, and his attempt is to improve the technique.

In all psychoses which go on to deterioration, there is a great increase of fatty substances in the cerebral cortex to be found mostly in the cells of the adventitia and the pia. He states that this can not come from a fatty degeneration of these cells themselves and hence must be the result of nervous tissue degeneration. He searches for the method by which this fat gets into these cells and has applied himself to the task of determining the nature of the intermediary degeneration products which lie between the normal brain tissue and this fat which he considers an end product. In this search for intermediary products he has described many things which exist as granules in pathological glia cells.

Besides its role of supporting substance for the nervous tissue, the glia has an important function in conveying nutrition to the nervous tissue, so that Alzheimer raises the question whether these peculiar substances he describes are all degeneration products. It is possible they may be products of a disturbed metabolism of the nervous substance. He offers his paper more as a description of what he has found than as an attempt to determine the nature of the substances.

II. METHODS OF INVESTIGATION

Here he gives details of ten different methods of staining glia cells and their inclusion bodies.

III. THE NORMAL STRUCTURE OF THE NEUROGLIA

Weigert first worked on the glia, giving most of his interest to the glia fibers which his method showed beautifully. He considered the fibers to be always intracellular, a view which today can not be upheld.

Held[4] later studied the protoplasmic processes of the glia cells and found that they anastomosed with each other to form a more or less loose reticulum—the supporting substance of the central nervous system. Besides this syncytium, Held also described the glia membranes which separate the central nervous system from the pia and surround all the blood vessels,—membrana neurogliar superficialis and perivascularis.

The glia reticulum does not exist in all parts of the central nervous system. Thus, it is more or less lacking in the deeper layers of the cortex and in the gray matter of the cord.

Many glia cells have very long processes (Pl. 28, 1 a & b). Others are more rounded and have granular cell bodies (Pl. 28, 1 e, f, g).

It has not as yet been determined whether all the glia cells of the central nervous system belong to the reticulum or whether some of the cells may exist independently. We know that in severe degenerative processes of the nervous tissue many glia elements do exist independently, take on peculiar forms, assimilate degeneration products and then degenerate. If normally they all belong to the syncytial network they must, under these conditions, be released from it.

Held believed that the glia membrane around the vessels served as a filter for nutritive fluids, but Alzheimer believes that cells may pass through it.

The author then discusses at length whether there normally exists a perivascular space around the brain vessels and concludes that it is a matter of great doubt in normal brains. However, under pathological conditions, there can be no doubt that such spaces exist through the disappearance of the membrana perivascularis and in these perivascular spaces a considerable part of the degenerative processes of nervous tissue takes place. These spaces exist in the nervous tissue and they contain exclusively glia cells and never mesodermal elements. Alzheimer considers that this speaks against the preëxistence of perivascular spaces.

IV. THE PATHOLOGICAL CHANGES IN NEUROGLIA IN SEVERE ACUTE DISEASES OF THE CENTRAL NERVOUS SYSTEM

A. *The Amœboid Glia Cell and its Different Forms.*—Together with the appearance of amœboid glia cells, many forms of degenerative glia cells appear.

Amœboid cells in the white matter differ from those in the gray matter.

Alzheimer summarizes his findings as follows: We find in acute and subacute diseases of the central nervous system, especially in the cerebral cortex, pathological glia elements which usually rapidly disappear and never form glia fibers. Their usual form, especially those in the white matter, is amœboid. In the cortex, other forms are to be seen which are not so clearly amœboid. All these cells possess in common numerous granules, named by the author, fuchsinophile granules, light green granules, lipoid bodies and cysts, methyl blue granules and fibrinoid granules.

B. *Vascular Changes which Accompany the Appearance of Amœboid Glia Cells.*—Wherever amœboid glia cells appear in large numbers, many kinds of peculiar material are seen around the vessels in the perivascular spaces. A part of these masses consist of coagulation material from pathological tissue fluids. Besides these one finds amœboid glia cells, some fresh and others with signs of degeneration, in these perivascular spaces. These spaces are formed by a disappearance of the membrana limitans glial and are not artificial shrinkage spaces. When the above described changes exist, many cells of the adventitia and of the adventitial lymph spaces undergo fat degeneration.

C. *The Pia and the Degenerative Processes in Nervous Tissue.*—With the changes in the nervous tissue which manifest themselves by the appearance of amœboid glia cells, there go hand in hand also changes in the pia. This is shown in the disposition of various extracellular material and an increase of cellular elements with fat degeneration of the same.

D. *The "Fill Bodies" and their Relation to the Glia.*—Besides the degeneration products in the glia, Alzheimer also describes certain extracellular bodies to which he has given the name "Füllkörperchen." They are seen usually in diseases where amœboid glia cells exist in considerable numbers. These bodies he describes as being small or large, rounded or angular, sometimes scattered in the nervous tissue and at others collected into groups, when they make a mosaic-like appearance. In the early stages they stain deeply with light green, methyl blue and Molony's hæmatoxylin. Later they stain less intensely and still later appear to dissolve. Alzheimer believes they represent degeneration products of pathologically changed glia structures.

E. *The Biological Significance of Amœboid Glia Cells, the Circumstances of their Appearance and their Relation to Degeneration of Nervous Tissue.*—After a lengthy review of the degenerations of nervous tissue the author summarizes his observations as follows: The amœboid glia cells have nothing to do with the supporting reticulum. They do not form fibers. They exist only where nervous tissue is degenerating, as though the presence of degeneration products acted as a stimulus for their formation. They do not take up degenerated nervous tissue as such, but assist in the process of making this matter fluid. Hence Alzheimer believes that the amœboid glia cells assimilate the materials which result from the degeneration of nerve-cells and other nervous tissue. At the height of their development they rapidly undergo regressive changes, form various kinds of granules and rapidly degenerate. The degeneration products of these cells get into the lymph and perivascular spaces, in part in fluid state again, and are taken up by the mesodermal cells and changed into lipoid matter. Thus, the amœboid glia cells clear the nervous tissue of degeneration products, change these products to substances less dangerous to ectodermal tissue and finally transport them to the mesodermal tissue where they are eliminated.

VARIOUS DEGENERATION PRODUCTS

V. GRANULES—REICH[7]—PROTAGON.

First described by Reich[8] in 1907 in the cells of the sheath of Schwann of peripheral nerves. Alzheimer had previously seen a similar structure in glia cells and had termed it "basophile-metachromatic substance." This substance occurs only in the human brain. Alzheimer has never been able to observe it in any other vertebrate's brain or peripheral nerves. He agrees with Reich's view that the Schwann cells fulfill the same function in the peripheral nervous system that the glia cells do in the central nervous system.

VI. SIMPLE BASOPHILIC DEGENERATION MATERIAL.

VII. EXTRACELLULAR DEGENERATION PRODUCTS.

These have been known longer than intracellular ones and are less important.

Corpora amylacea—nature unknown. Obersteiner[6] thought they arose from axis cylinders, and others from the glia. Alzheimer doubts both of these assumptions. He considers they may consist of formed filtration products which are unable to penetrate the membrana from the fact that they tend to gather on the brain surface and around the larger vessels, always on the neural side of the membrana limitans glial.

Glycogen—Casamajor.[2]

Senile Placques—Fischer.[3]

VIII. SPECIAL DEGENERATION PRODUCTS IN CERTAIN DISEASES.

Amaurotic Family Idiocy.

Warren-Tay-Sachs. Form.

Spielmeyer-Vogt. Form.

Peculiar Stuff in Ganglion Cells.

Alzheimer discusses the staining characters of this material which are different from the granules he has previously described.

IX. THE DIFFERENT FORMS OF DEGENERATION IN NERVOUS TISSUE.

The author describes four types of nervous tissue degeneration:

1. Where nervous and neuroglia tissue are both destroyed as from trauma, hemorrhage or softening.

Here the work of removing the dead tissue falls principally on the mesodermal elements. New vessels bud in from the edges of the lesion. Adventitial cells separate themselves from the walls of these vessels, wander with the surrounding matter, take up the dead material, and convert it into fat, becoming thereby fat granular cells. These then disappear, and the fatty matter they contain is taken up by the blood and lymph. In the meantime the glia cells on the lesion margin rapidly form fibers which completely encapsulate the lesion. In the end stage the lesion consists of new blood vessels, scar tissue and a glia capsule.

2. When the degeneration affects only nerve-fibers (the white matter).

Here the glia does most of the cleaning-up work. The medullary sheaths break up into fat droplets. Wandering glia cells take up this fat and become gliogenous fat granular cells in contradistinction to the mesodermal ones mentioned above. These cells then wander toward the blood vessels and out to the pia. Here they break up and the fatty matter is taken up by mesodermal cells and later is seen lying in the adventitia and the adventitial lymph spaces. When this cleaning up process is at its height, other glia cells begin to form fibers which stretch through the tissue till at the end stage the nerve-tissue is replaced by a neuroglia scar. This is the type of degeneration seen in Tabes and other tract system diseases, Multiple Sclerosis and in pyramidal tracts after hemiplegic strokes, etc.

3. When the degeneration process is slower and of less severity.

Here the amœboid glia cells do the work of cleaning up. The degeneration products do not consist primarily of fat, but are seen in the amœboid glia cells in the form of various granules. These cells wander out into the perivascular spaces where they break up into fat and fat-like granules. These are in turn taken up by the adventitial cells and all converted into fat which passes off in the lymph. Increase of glia fibers here follows the cleaning-up process and does not accompany it as in Type 2, and exists as an increase of glia fibers in the cortex, not a glia scar. This type of degeneration is seen in Dementia Precox, Epilepsy, General Paresis, and other diseases which have stormy episodes.

4. Here the process is of still less severity. The glia does the cleaning-up work but amœboid glia cells are not seen. Ordinary glia cells take up the degeneration products and hold them in sac-like cysts, or else the whole cell body becomes full of fatty bodies. Sometimes these granules are cast off by the cells. The adventitial cells are also full of fat. This type of degeneration is found in many brains in chronic diseases. In a very mild form, it is to be seen in practically every brain of adult human beings and especially in the aged. Alzheimer says that if we are to look upon the presence of fatty material in ganglion glia and adventitial cells as pathological, then there is no such thing as a normal human brain.

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